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VETERINARY MEDICINE

JANUARY 1943

CONTENTS

Veterinary Progress, Policies and Comment 1-7

Cattle Population of India, 1; Shark Carcass Has Many Uses, 1; Tyrothricin in Mastoid Operation, 1; Lead Line at Camp Grant, 2; Cavalry Suited to Various Tasks, 2; War and Veterinary Progress, 2; Help the Cause, 3; Stamp Out This Saboteur, 3; Laboratory of Animal Pathology, M.S.C., 4; Britain's Police Horses, 5; Increase in Price of Work Horses, 5; Penny Wise Pound Foolish Legislation, 5; Veterinary Officer a Bomber Pilot, 6; Commissioning of Veterinarians, 6; Mice Inoculated with Trypanosomes, 6; Virgin Goats Made to Lactate, 6; Revival of Bipp, 7; Inhibitory Action of Bipp, 7; Repellents, 7; Derris Root in Scabies, 7.

Seasonal Anthelmintic Treatment of Horses 8

Gastralgia Due to Bot Infestation in Horse 12

Bovine Mastitis 14

Isolation of Western Encephalomyelitis and Hog-Cholera Viruses from Supposedly Hog-Cholera Immune Hogs .. 22

Sulfathiazole for Colds in Chickens 26

Autobiography of a Veterinary Practitioner 29

Clinical Coccidiosis in Puppies 32

Technic for Handling a Self-Sucking Cow 34

Record Your Experiences 35

Book Reviews 36-40

Ambassadors in White, 36; Breeding and Improvement of Farm Animals, 37; The Springer Spaniel, 38; Veterinary Surgery Notes, 38; The Officers' Guide, 39; The Doctors Mayo, 40.

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Muskrat flesh is sold for food in some city markets under the name of marsh rabbit.

* * * *

That vitamin A in massive doses counteracts high blood pressure, caused by lack of function of the kidneys, has been demonstrated by the University of Illinois College of Medicine.

* * * *

At the beginning of recorded history, veterinary medicine was already a distinct art in the hands of specialists. As to how this came about, or how the veterinary personnel was trained, history is wholly silent. The laws of Hammurabi (2100 B.C.) provided rigid regulations for the conduct of veterinary practice. They could not have been observed except by carefully trained men.

* * * *

Tyrothricin applied to ulcers resulted in sterilization and healing if the local infection was caused by *Streptococcus haemolyticus, Staphylococcus aureus* or *Streptococcus faecalis* and encouraging results were obtained when it was applied to mastoid cavities following mastoid operations, Charles H. Rammelkamp, M.D., Boston, reports in a recent issue of *War Medicine*.

Elephants were the tanks of ancient armies.

* * * *

In 1940 more than three million cattle in India were vaccinated against rinderpest, 150,000 against hemorrhagic septicemia and 21,000 against blackleg.

* * * *

It is the duty of veterinarians to conduct their private practices in an ethical, professional manner and to refrain from acts of commission or omission calculated to bring discredit upon the veterinary profession or undermine the confidence of the public in scientific veterinary medicine.

* * * *

Shark-fishing off the south section of the Florida east coast, has become a booming business. Heretofore little interest was taken in shark-fishing but since Norwegian cod liver oil is unobtainable and a single shark liver has been found to contain anywhere from two to 25 gallons of oil, a flourishing industry has been developed. In addition to liver oil with a large vitamin A content, other parts of the carcass are being utilized in leather, dog and poultry food, fertilizer, Chinese soup, souvenirs, etc., so there is now very little waste from a shark.

Lead Line at Camp Grant, Illinois

Veterinary Technicians School, Camp Grant, Illinois, demonstrating the operation of a veterinary lead line. The officers in the center foreground are, left to right: Col. O. H. Dixon, Jr., VC, head of the school; Maj. Normon Stoner, VC, senior instructor and Lt. Francis L. Riley, VC, instructor. The lead line is designed for evacuating animal casualties over short distances. With it, four mounted men can evacuate 20 animals at a time. The field of usefulness of the lead line as an apparatus for evacuation is quite limited

Cavalry Suited for Varied Tasks

Cavalry can cope with varied situations. It can ferret out anti-tank units waiting in ambush. It can protest the flanks of the motorized infantry in battles and form a link with the slower moving marching divisions. It can cope with guerrilla warfare. It is well suited to hunt down parachutists. It has the flexibility and mobility which enables it to fill in all the weak gaps. It has been shown that the tank is not very dangerous for the cavalryman if he can handle hand grenades, mortars and automatic arms, and can fight on foot as well as on horseback.—*Nat'l. Horseman.*

War and Veterinary Progress

A hundred and seventy-eight years ago a scientific veterinary service was established in France and, within a decade, in every important country of continental Europe. Great Britain inaugurated its veterinary service 30 years after that of France was founded. The precise reason for the institution of these services was to assure a food supply for the great armies which the nations desired to put into the field. Armies had reached a size where they could not feed themselves by the pillage of occupied territory. Thus the object of the veterinary services was the promotion of bigger and better wars.

The demand for saddle horses well broken to drive, is reported to be increasing with prices ranging from $75 to $300 per head.

Because of difficulty in obtaining vehicles, few work horses have been sold in cities.

Help the Cause

For what cause is this nation arming if not on behalf of the men, women and children who compose it? Their physical fitness, their freedom from preventable disease, their morale or mental stamina, will determine almost entirely the effectiveness of all other defense efforts. Important in the easy days of peace without a cloud on the horizon, it is urgent now that the people of this nation be physically tough, mentally sane and morally sound.—*Thomas Parran.*

* * * *

Stamp Out This Saboteur*

A saboteur, *Mycobacterium tuberculosis,* threatens our war industries. He does not sneak in at the factory gate to light a damaging fire or blow up the plant with hidden dynamite and neither armed guard nor the F B I itself can detect or stop him. But every loyal American can help to detect and stop this saboteur by buying Christmas Seals.

In the four years, 1937-1940, the *M. tuberculosis,* killed more Americans than died as the result of action in all the wars our country fought up to December 7, 1941.

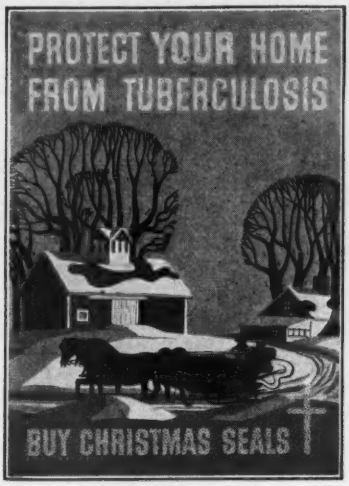
This germ is especially detrimental to our peacetime army of industrial workers, including young girls and women who by thousands are engaging in war industry.

Since we are at war we need the utmost in health and strength to fight on to victory, and during wartime, this disease threatens more than ever.

Tuberculosis has been called the Fifth Horseman of the Apocalypse. Riding in on the increased tempo of longer working hours, stress and strain, poor nutrition and crowded living quarters, it takes a greatly augmented toll of health and life. During the World War of 1914-1918, the death rate of this disease was increased 50% or more in some of the warring nations and even doubled in certain cities of Europe.

The difficulty in fighting this disease enemy is that, like all other saboteurs, he works stealthily and under cover. Unless he is detected and brought under control he will hinder the flow of guns and planes and shells to our men on the firing line and slow down the production of ships to carry the men and their supplies.

If every worker is given an X-ray examination of his chest before he starts to work the development of great numbers of tuberculosis cases can be halted. This examination reveals the presence of tuberculosis long before the patient is sick, long before any symptoms make him or his doctor suspicious. The Army and the Navy have used chest X-ray examinations to eliminate tuberculosis cases from among our fighting forces. The National Tuberculosis Association recommends that the same measures be taken to control tuberculosis in our war industrial army and among the civilians.



Every time you stamp Christmas Seals on a letter or gift package you are helping to stamp out the master saboteur of our war industries. Ever since 1907 the Christmas Seal Sale which the public has been supporting has provided national and local tuberculosis associations with munitions of war against this disease enemy. Now, more than ever, with tuberculosis deaths already mounting in Europe as a result of the war, your help is needed in the fight to prevent and control tuberculosis in America.

*Jane Stafford, National Tuberculosis Association.

Laboratory of Animal Pathology, M. S. C.

Illustrations Nos. 1 and 2, show the use of the microscope in diagnosis, as demonstrated at Michigan State College during the annual Veterinary Short Course.

Fecal examinations are made to determine the presence of parasites. The man pictured at the microscope is Doctor Sholl, assistant animal pathologist, Division of Veterinary Medicine. The interested observer is John W. Patton. In No. 3 C. G. Jensen, Petosky, Mich., is drawing a blood sample from the jugular vein of a Great Dane, using a 16-gauge needle. An assistant holds the dog's head elevated and turned, distending the vein with his thumb.

These blood samples are collected in tubes containing anticoagulant, to be used for making hemoglobin estimations, red and white blood cell counts and blood chemistry determinations. For the latter glucose, creatinine, and non-protein nitrogen are used. They are of particular value in diagnosing diabetes and nephritis. In diagnosis of anemia, leucocytosis, leucemias and malignant neoplasms, hemoglobin determination, blood cell and leucocyte counts are of value.

No. 4 illustrates catheterization to obtain a sample of urine. Doctor Sholl furnishes the necessary restraint. Wade O. Brinker and John W. Patton are in the background. In several cases an unsuspected urolith has been discovered. At such times the catheter should not be forced past the obstruction as it may lacerate the urethral mucous membrane.

In this laboratory many specimens, including skin scrapings for mange are examined for parasites and fungus. Examinations of the sediment of urine for the presence of abnormal constituents and tests for glucose, albumin and indican are routinely made to aid in diagnosis. They are also made for the identification of lice, determination of the potency of semen, for the presence of trichomonads in vaginal secretions, etc.

During the past year more than 1500 clinical laboratory examinations of various types were made by the pathological laboratory of the veterinary clinic at Michigan State College. This represented 2,342 individual tests. The number of examinations made increases year by year.

Turn your geldings or mules into good young mares that you can work and breed. —*Wayne Dinsmore.*

Britain's Police Horses

Since the horse is probably the most timid animal, it is astonishing that London's 250 or more police horses have gone through many devastating raids on the capital and not a single one has been hurt. In the training of these animals for service the most difficult part is to get them to face fire and smoke. It usually takes six months to train a horse for police duty.

January 1, 1942, mules of all ages in the United States were valued by the U. S. Dept. of Agriculture at $107 per head; horses at $65.00 per head. The average farm value for the 10 years, 1931 to 1940 inclusive, all ages included, was $98.00 for mules and $76.00 per head for horses. This difference in price, favoring mules, plus the fact that mules are more readily sold as foals, yearlings or 2-year-olds than horses, makes it advisable where a good jack is available to raise mules instead of work horses.

According to the Horse and Mule Association the better class of work animals have advanced in price at least $10.00 per head in the past 60 days. Small grade draft mares, from four to six years of age, standing 15.2 to 15 3, weighing 1200 to 1300 pounds, of good type, sound, and green broken to work, are selling at from $170 to $250 per pair; geldings of the same type, but larger, weighing about 1500 pounds each, $250 to $300; spans of mules about 15.3, weighing 1400 pounds each, $350 for jacks and $400 to $550 for mares of the same size and quality. A few heavy draft horse teams 2000 pounds each and up, of good type, quality and finish are bringing around $400 to $450 for shipment to Maine potato fields, but must be choice or will not sell. Top mare mules, weighing from 1200 to 1400 pounds, are going to tobacco growers in the Carolinas, at $450 to $500 a pair—a few at $550,—so that through all classes, mules are bringing from $50.00 to $100.00 per span more than horses suitable for the same work.

Outbreaks of hog cholera appeared in many sections of the State (North Dakota), resulting in heavy losses. A great many of these outbreaks were due to farmers vaccinating their own hogs.—*Rept. State Veterinarian.*

Peanuts for Hog Fattening

In order to aid in war shortage of oils for fattening hogs the farmers of America were requested to raise 5,000,000 acres of peanuts during 1942. This is more than twice as much as was produced in 1941.

The pork product when peanut oil is used differs only in that the fat is of a softer consistency. The bacon from hogs fattened on peanuts is thoroughly unsatisfactory, because of softness and loss of weight.

Penny Wise Pound Foolish Legislators

During the legislative session of 1935, House Bill No. 112, an act regulating the purchase, use, administration and handling of hog-cholera virus, became a law. This act permits farmers to vaccinate their own hogs. At the hearing before the Livestock Committee your Executive Officer at that time explained the dangers of legislation of this kind and advised that if the bill passed hog-cholera would spread to all counties of the State within a few years, instead of being confined to a few of the southeastern counties.

During the past summer and fall we have seen this prediction come true—the State having suffered the heaviest hog loss it has ever known. Most of the outbreaks can be traced to vaccination by laymen, selling of hogs a few days after double treatment or buying untreated hogs and placing them with treated hogs.

It seems too bad that legislators can be so shortsighted as to permit destructive legislation of this type to pass. This Board (Livestock Sanitary Board) is helpless to cope with a condition of this kind with the limited appropriation given it, and, in any case, hog-cholera cannot be controlled in the face of unrestricted use of hog-cholera virus.—*From the Annual Report of the State Veterinarian of North Dakota.*

1st Lieut. Warren H. Smeltzer, V. C., formerly in practice at Mendota, Ill., is now a student officer pilot in the advanced flying school at Ellington Field, Texas. Upon graduating from Ellington, he will trade his "V" and Caduceus for Wings

School for Medical Technicians

The School for Army Medical Technicians, William Beaumont General Hospital, El Paso, Texas, is designed to furnish highly-trained technicians.

The so-called specialty groups, which are laboratory, pharmacy, dental, x-ray and veterinary, require three months to complete the courses offered. There are prerequisites as regards educational background.

There are two classes of veterinary technicians, medical and surgical, and meat and dairy inspection assistants. The former must be familiar with the care and use of instruments and apparatus of the veterinary dispensary; be competent to take temperature, pulse and respiration of the animals; to care for and maintain a veterinary ward; prepare and feed special rations; administer medicines, orally and hyperdermically; apply bandages and dress wounds; give enemas; be proficient in methods of restraint of animals; prepare and care for all records of the clinic and assist at operations. The veterinary technicians in the meat and dairy inspection have as many and varied tasks as have those in the medical and surgery group.

The Commissioning of Veterinarians

The directing board of the Procurement and Assignment Service, Washington, D. C., recently requested information from the Surgeon General's Office about its policy concerning the commissioning of doctors of veterinary medicine. It seemed to the board that valuable veterinary manpower was being lost through the induction of veterinarians as enlisted men and their subsequent commissioning through routine military channels.

The board asked the Surgeon General's Office if it would not be possible for veterinarians to apply for commissions on receiving their orders for induction. In reply, the Surgeon General's Office gave the following information:

1. Routine commissioning of additional veterinarians in the Veterinary Corps was suspended two years ago.

2. Veterinarians now commissioned are in excess of the current needs and the needs of the immediate future.

3. The conserving of veterinary manpower should be considered a function of Selective Service.

4. If considerable numbers of veterinarians are inducted as privates, the War Department will be unable to commission them soon for lack of position vacancies.

The directing board has recommended that all state veterinary chairmen immediately prepare surveys of the veterinary manpower and its distribution for their respective states in order that the need for the occupational deferment of veterinarians might be factually substantiated in requests to the local Selective Service boards for the deferment of veterinarians. The data included in these surveys should be sufficient to enable these boards to judge the essential character of the work of any veterinarian for whom deferment is sought on the ground of occupational essentiality. It is further suggested that state chairmen immediately communicate with the state director of Selective Service for a discussion of the problem. It is apparent that deferment in the future will depend on the decision of the Selective Service boards.

A Revival of Bipp

The *Pharmaceutical Journal* (London) observes: "Many chemists will recall the paste of bismuth subnitrate, iodoform and liquid paraffin which was so popular for dressing wounds during World War I. Bipp, as this combination is commonly called, was in fact in use before the introduction of the irrigation methods of Carrel and Dakin. It is significant that the paste has retained its popularity in some quarters in spite of the fact that its mode of action even now has not been fully explained; but it is even more significant that it has survived in spite of the high hopes that have been entertained for such newer antiseptics as the sulfonamides and penicillin. The logical inference is that Bipp does what is expected of it and doubtless will be in great demand in the civilian and military hospitals during the present conflict.

"One or two interesting observations were made regarding its position in a survey published by a current medical journal. An attempt to avoid the toxic effects of bismuth has been tried by substituting zinc oxide for bismuth subnitrate, and 'Zipp,' which was introduced ten years ago, is still undergoing trial. The second point which has been noted is that soft paraffin is not a satisfactory substitute for liquid paraffin in these pastes, as it does not adhere to the walls of the wound. This applies also to other preparations; sulfanilamide is said to be inert if mixed with an ointment base, whereas in liquid paraffin its full antiseptic action is exerted. These factors provide additional reasons for conserving supplies of liquid paraffin."—*The Veterinary Record.*

When there are no foreign bodies or necrotic tissues in the depth of the cavities of fistulas, abscesses and deep flesh wounds and when the horse is capable of being on duty there is an advantage in the use of disinfecting pastes (such as Bipp) as they are adherent and keep a continuous contact with the surfaces of the wound; they also have an inhibitory action upon secretion and prevent the spread of infection into the deeper layers of this tissue.

Repellent

Fly pests in Military Camps in Mesopotamia, have been controlled by using a mixture of half a pound of sodium arsenate and two and a half pounds of jaggery* in two and a half gallons of water. Strips of cloth kept on a tin like a roller towel dipped in this mixture and hung up with one end kept soaking in the mixture attracted flies even from a distance of 200 yards. This trap was modified and made simpler by using an empty ghee tin and the following mixture:—

Sugar, molasses or jaggery	1 lb.
Sodium arsenate	1 lb.
Water	10 gallons

This was found to be effective in Military Hospitals also.

Creosote oil sprayed inside mess halls and cloth soaked in this oil hung over the entrance is an efficient repellent and keeps a room practically free from flies.

Derris Root Effective in the Philippines Against Scabies and Ticks

Infusions of crushed derris roots in water at a concentration of 2.5% fresh roots or 1% dried roots, applied on each of two consecutive days, were effective against equine scabies. For bovine scabies a concentration of 3% fresh roots or 1-5% dry roots was required. The derris infusion did not damage the skin, hair or eyes of the treated animals.

Infusions, varying in concentration from 1.25-5% were found to be effective in killing ticks on cattle and horses. The smaller ticks died in 24 and the larger ones in 48 hours. To ensure the destruction of the ticks, the skin and hair had to be thoroughly saturated with the infusion. It is recommended in preference to arsenical solutions for swabbing or spraying.

---

* A sugar made from the jaggery palm.

Seasonal Anthelmintic Treatment of Horses

By C. E. HOWELL* and J. W. BRITTON**

The routine anthelmintic treatment of horses is an important problem. We are frequently asked, "How often should I worm my horses?" This is often difficult to answer. Except in weanlings and yearlings, the average horse, unless maintained under poor conditions, shows relatively few clinical symptoms from even moderately heavy strongyle infections. Of course, in extreme cases, the worm burden may become so excessive that clinical manifestations are apparent even under ideal conditions of management. The damage from strongylosis in mature horses is usually subtle and probably manifested chiefly by loss of resistance to disease, overwork, fatigue and inadequate rations. Sucklings, weanlings and yearlings, and occasionally two-year-olds, more often suffer from strongylosis as a primary disease. Clinical strongylosis is manifested in these animals by loss of foal flesh, diarrhea, stunting, emaciation and anemia with an occasional death from thrombo-embolic colic. A worm burden which would be pathogenic to a weanling, often can be tolerated by adult horses with no outward symptoms, provided feed and management are satisfactory. Many mares in excellent condition show strongyle egg counts over 4000 EPG (strongyle eggs per gram of feces)—a count almost invariably accompanied by symptoms in yearlings.

The University of California's Arabian Horse Ranch at Pomona offers an excellent opportunity to observe the course of clinical and subclinical parasitism in horses in a breeding establishment where routine anthelmintic treatment is desirable. The majority of the horses are pastured all year.

* Manager, University of California's Arabian Horse Ranch, Pomona.
** Division of Veterinary Science, University of California, Davis.

Objectives of Anthelmintic Treatment

To determine how frequently it is necessary to administer routine anthelmintic treatment, the ultimate objectives must be clearly understood. The authors have based the recommendations herein on three main objectives.

Striking "action" photo, called the "Three Graces," taken at the University of California Arabian Horse Ranch, Pomona

1. The cure of clinical cases of strongylosis.

2. Prevention of clinical strongylosis in sucklings, weanlings and yearlings.

3. Maintenance of the strongyle infection in mature horses below a point at which resistance to disease and adverse environmental factors are lowered.

The first objective is obviously necessary and concerns treatment for cure. The efficiency with which the second and third objectives are carried out as preventive meas-

ures will materially lessen or obviate need for the first objective.

Prevention of Strongylosis in Foals

It has become an established tradition, almost as deeply rooted in the minds of horse breeders as ninth day breeding, to worm foals early and often. The logic of this practice is difficult to understand. Baker, Salisbury and Britton[1, 2, 3] showed that strongyle eggs first appear in the feces of foals at from 95 to 120 days of age. Obviously the worms responsible for these eggs were acquired early in life but have remained embedded in the intestinal mucosa or other inaccessible portions of the body, safely hidden from the action of anthelmintics, but producing real tissue damage. The eggs of *Trichostrongylus axei* commonly appear when the foal is from 30 to 33 days of age and are doubtess the cause of much confusion concerning the proper time to treat foals for worm infestation. These eggs are indistinguishable from those of the strongyles although the infective larvae reared from a fecal culture are readily identified. There is doubtless a short non-egglaying period when the strongyles, as immature adults, first reach the intestines but practically always the appearance of eggs in the feces signifies the first advent of the adult worms in the colon.

TABLE 1. THE AVERAGE STRONGYLE EGG COUNTS (EPG) OF FOALS FROM 1 TO 24 MONTHS OF AGE

Age in months	No. in group	Ave. EPG	Range in EPG	No. in group over 1000 EPG
1	12	0	0 - 0	0
2	14	0	0 - 0	0
3	15	0	0 - 0	0
4	8	50	0 - 200	0
5	16	300	100 - 900	0
6	14	555	200 - 1100	1
7	16	570	0 - 1200	1
8	15	680	200 - 1200	2
9	10	380	200 - 1400	2
10	17	900	200 - 1200	8
11	10	890	400 - 1800	3
12	16	1725	400 - 2500	12
13	4	950	700 - 1300	2
14	8	1300	800 - 3000	7
15	8	1635	600 - 2200	6
16	8	1360	700 - 2000	4
17	5	1650	500 - 4500	5
18	5	1400	600 - 2500	4
19	3	1700	1000 - 2400	3
20	3	1400	1000 - 2200	3
21	3	2200	1200 - 2600	3
22	1	2000	2000	1
23	5	2500	1500 - 3000	5
24	5	2500	1500 - 3400	5

During the first few months of life, foals depend chiefly on the dam's milk for nourishment but as they grow older, the amount of grazing steadily increases. This means that the parasitic infection acquired during the suckling period is picked up chiefly in the last four or five months (assuming a weaning time of seven or eight months). Counting a minimum encysted larval stage in the intestines as 90 days, strongyle eggs do not appear in the feces in any appreciable numbers until weaning time. This fact was emphasized by Baker, Salisbury and Britton[1, 2, 3] working with Percheron and Belgian foals. They found that nine months represented the time at which a threatening number of eggs appeared in the feces.

Strongyle egg counts and fecal cultures were made every six to eight weeks on 50 Arabian foals at the University of California's Arabian Horse ranch and the results (Table 1) amply confirm the findings obtained with the heavier breeds.

Athough the strongyle egg count does not attain serious proportions until the ninth or tenth month, the larval forms which emerge as adults around the 11th or 14th month are producing damaging pathological lesions in the intestinal mucosa. Consequently even though treatment is administered, only half of the necessary preventive measures have been taken. Obviously the ideal is to prevent infection of the foals during the early months of life and it is practical to do this by routine treatment of the brood mares shortly after foaling and again in four to five months. Both the treatment of the foaling mare and of the foal at weaning can be accomplished easily and safely by administering small repeated doses of phenothiazine in the grain usually fed at this time.

The efficacy of small repeated doses of phenothiazine was suggested by the authors in an earlier paper[4] and further trials with this method of administration have confirmed those results. Beginning July 6, 1941, nine head of Arab stallions and mares with an average pretreatment EPG of 1433 were given 5gm of phenothiazine morning and night for three days. By January 15, 1942 the average count had risen to only

300 EPG and by June 20 it was 1000 EPG. Nine head of stallions and mares were given a single 30gm dose of phenothiazine on July 6, 1941 at which time they had an average count of 1670 EPG. The January 15, 1942 and June 20 counts on these horses were 400 and 1000 EPG respectively. Two yearling stallions with an average count of 2100 EPG were given 6gm of phenothiazine a day for five days beginning on December 14, 1941 and by May 1, 1942 the average count of these two stallions had risen to 250 EPG. Two untreated controls of the same age and in the same environment showed an average rise in the count from 800 EPG on December 14 to 1850 EPG on May 1. Obviously the results were entirely satisfactory. Eight yearling stallions with an average pretreatment count of 1450 EPG were given 1gm of phenothiazine daily from April 29, 1942 to May 13, 1942 inclusive, a total of 15gm. May 20 the egg count was negative and by June 20 it had risen to 400 EPG. Eight untreated controls in the same pasture and of the same age had had an average count of 1000 EPG on April 29 and 1700 EPG on June 20. These results indicate that it is possible to use a smaller total dose of phenothiazine when it is administered in small repeated doses and thereby eliminate many toxic reactions. The authors believe it to be the method of choice for treating mares and weanlings.

In order to maintain the maximum of growth and development in foals during the first 18 months of life, during which time parasitic infestation is most important, it is recommended that foaling mares be treated routinely and the treatment be repeated in four to five months. The foals should be wormed first at weaning time (seven to eight months of age) and again when they are from 11 to 14 months of age. It has been observed that even moderately infected weanlings and yearlings which are rough coated, lacking vigor and considered to be clinically parasitised, develop, under adequate conditions of feed and management, into good two-year-olds without the benefit of anthelmintic treatment. The advantages of routine treatment for the prevention of strongylosis in foals are, that the foals make a more satisfactory growth, are always in good condition for sale and develop into stronger, more vigorous two-year-olds than if they had been allowed to combat the effects of parasitism unaided.

TABLE 2. THE RISE IN EGG COUNT OF 62 HORSES FOLLOWING TREATMENT WITH PHENOTHIAZINE

Age group years	No.	Date treated	12-1-41	No. with strongyle EPG over 1000 On: 1-15-42	3-1-42	4-21-42	6-20-42
1 - 4	26	7-6-41	10=38.5%	16=61%	17=65%	23=90%	26=100%
5 - 10	14	7-6-41	1= 7%	3=21%	4=30%	6=43%	8=60%
over 10	11	7-6-41	0	0	0	0	4=36%
1 - 4	3	8-7-41	0	2=66%	2=66%	2=66%	3=100%
5 - 10	4	8-7-41	0	2=50%	2=50%	2=50%	3=75%
over 10	4	8-7-41	0	0	0	0	1=25%

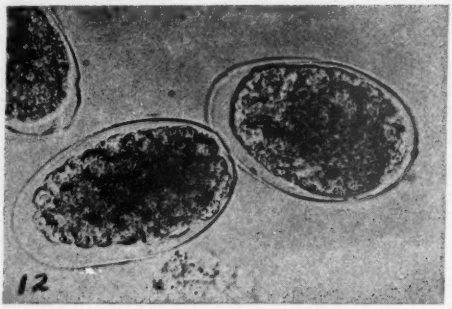
Routine Treatment of Mature Horses

In order to keep the parasitic infestation in adult horses below the point at which clinical manifestations are liable to appear under adverse conditions, routine treatment is necessary. Chiefly through observation, an egg count of 1000 was considered to be the maximum worm burden which a horse could handle and the degree of infestation which should indicate treatment.

Sixty-two Purebred Arabians were used in a study to determine the frequency of treatment necessary to maintain the strongyle infestation below 1000 EPG. A few of the horses were stabled but the majority were pastured. No significant differences could be found in the rate of pickup of infection between stabled and pastured horses, hence the herd will be considered as a unit. One group was treated with 30gm of phenothiazine on July 6, 1941 and a second group similarly treated on August 7, 1941. Egg counts were then made of the feces of all horses every four to six weeks for a year. The results are above in Table 2.

Three untreated controls in the one to four age group showed a steady increase in egg count up to March and April when there was a slight decline. Seven controls in the oldest group showed no important deviations from the original count, bearing out the statement made by Britton[5] that

middle aged and older horses frequently lose strongyle infestation as rapidly as they acquire new worms so that the egg count remains fairly stationary.



No. 12. Eggs of *Strongylus vulgaris*, x400.—*Benbrook*

Twelve of the group of animals one to four years of age were retreated on December 1, 1941 and six were retreated on January 15, 1942. Of the twelve. six required a third treatment on March 1; eight were over 1000 EPG on April 21 and all were in need of a third treatment on June 20. Of the six retreated on January 15, three needed a third treatment on April 21 and all were over 1000 EPG on June 20. Evidently environmental conditions favorable to larval development in Pomona are just the reverse of those reported by Baker, Salisbury and Britton[1, 2, 3] in Ithaca, N. Y. Apparently under Southern Californian conditions the greatest pickup of infective larvae occurs during the warm rainy season in the winter and spring and in the dry summer and fall months become relatively lethal to the eggs and young larvae on pasture.

Recommended Treatment

The following recommendations for routine anthelmintic treatment of adult horses are based on the figures for the most susceptible 40 or 50% of the horses. Horses from one to four years of age should be treated in July, December and April. Horses from five to 10 years of age need two treatments (July and January) a year and horses over 10 years of age should be treated once a year preferably in July or August.

It is not intended that the recommendations suggested in this paper be followed in every locality. Obviously environment is the limiting factor, although the egg count at which it is deemed advisable to worm horses may also vary with the breed. Baker, Salisbury and Britton,[1, 2, 3] for example, showed that the Percherons at Cornell University were definitely more susceptible to strongyle infection than the Belgians. In limited observations, the Arabians proved far less likely to acquire the same number of adult strongyles per infective larvae presented than did several Percherons maintained on the same pastures. The recommendations for routine anthelmintic treatment herein reported are meant therefore to apply chiefly to horses maintained under conditions similar to those at the University of California's Arabian Horse Ranch.

Summary

1. Obvious clinical cases of strongylosis should be treated at once.

2. Brood mares should be wormed with small repeated doses of phenothiazine as soon after foaling as it is safe to give a grain ration in which the drug can be administered.

3. Foals should be wormed, when they are weaned with small repeated doses of phenothiazine and again when they are 11 to 14 months of age.

4. Horses from one to four years of age should be treated in July, December and April.

5. Horses from five to 10 years of age should be treated in July and January.

6. Horses over 10 years of age should be treated in July or August of each year.

Literature

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Gastralgia in a Horse Due to Bot Infestation

By PAUL C. UNDERWOOD[1] and G. DIKMANS[2]

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Recently, one of the writers (P.C.U.) encountered a case of gastric colic in a horse which subsequently terminated in a ruptured stomach and death of the animal. Autopsy revealed a heavy infestation of bot-fly larvae to be the immediate cause.

The horse, a sixteen-hundred-pound, eight-year-old gelding, employed at the Dairy Experiment Station at Beltsville, Md., as a work animal, sickened about midnight, Aug. 3, 1942. At this time somewhat muffled thumping noises were heard near the horse barn but no investigation was made. At four o'clock in the morning of Aug. 4, the herdsman noticed that the animal was showing evidence of colic. At 8:00 a.m. the following clinical picture was found.

The horse was standing, its body covered with dirt from rolling, eyes half closed, and showing all evidence of toxic dejection. Occasionally the animal turned its head to the right side and bit at the skin. This action was followed by a slight gurgling sound, and as the head was brought back to its natural position a thin stream of fluid containing crushed oats and hulls would trickle from the nostrils, forming a small puddle at the animal's front feet. Several such accumulations were observed in the barn lot. Further examination of the horse disclosed a rapid, thready pulse, temperature of 103.0° F., abdominal muscles rigid, and peristalsis absent on immediate ausculation.

Following this examination a diagnosis of stomach rupture was made, and an anodyne administered to relieve pain until death should occur. The animal died two hours later, at 10:00 a.m., and the carcass was removed immediately to the autopsy room.

Autopsy Findings

On opening the abdomen, stomach contents were found strewn over the viscera and peritoneum. The visceral and parietal peritoneum contained numerous large diffusely reddened areas indicative of early peritonitis. The stomach when first observed was practically empty of ingesta, and had a longitudinal tear twelve inches in length along the lateral wall of the muscular coat. The actual complete rupture of all stomach layers was ten inches in length. Several blood clots were noted in the ingesta near the stomach and clinging to the torn edges of the stomach proper. The stomach was removed and washed carefully, to avoid detachment of the bot larvae which could be observed clearly as a tightly formed mass attached to the stomach mucosa of both the cardiac and pyloric sections.

a *b*

Horse botfly, *Gasterophilus equi*,[1]
a lateral view *b* dorsal view

Eggs of this gadfly are taken into the horse's stomach with the feed and there the larvae (bots) develop. The oxbot, *Hypoderma bovis* lives under the cuticle of the ox, while the bot of sheep *Oestrus ovis* chooses to live in the frontal sinus of that animal

Nearly 200 bot larvae had been present in the stomach, as it was discovered later that in addition to those found attached to the stomach mucosa, a considerable number had become detached and dispersed among the viscera of the peritoneal cavity. None were observed in the small intestine. The larvae present in the stomach were uniform in size, appearing to be one-half to two-thirds grown.

This case shows rather clearly the damage which can be inflicted by certain parasites in their attack on a vulnerable host. It is quite clear that in this case the bot larvae proved to be such a source of nervous irritation to the stomach that gastralgia, followed by rupture and death, resulted.

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[1] From *Century Dictionary*, courtesy *D. Appleton-Century Co.*, New York.

The uniformly large size of the larvae found indicates that the animal had been infested for some time. This may be explained by the fact that this portion of Maryland, along with many other sections of the eastern seaboard, has comparatively early spring weather. Such weather favors the early emergence of the flies and the early infestation with bot-fly larvae. Such early infestations may be expected to occur in other sections with comparable weather conditions.

The present case would probably be found to be not unusual if the causes of gastric disturbances in horses in summer could be determined accurately, but many cases of gastralgia are relieved by symptomatic treatment without the underlying cause being ascertained. It is believed that bot-fly larvae may be the predisposing cause in many cases of gastric illness occurring in summer, especially in areas with early spring weather.

Stomach of a horse that died at the Dairy Experiment Station at Beltsville, Md. Gastralgia, followed by rupture of the stomach, was the result of heavy infestation by bots (maggots)

This case also indicates that in these areas it might be advisable to administer bot treatment during the summer, that is, in late July or early August. This treatment would eliminate the bots then present in the stomach and prevent much damage and loss of time due to illness caused by them.

Treatment of Bovine Mastitis*

By VILO T. ROSE,
Elkton, Kentucky

WITH bovine tuberculosis and brucellosis of cattle well in hand veterinarians are confronted with another disease, mastitis, which has almost completely baffled the efforts of research workers. For years this condition has been considered primarily infectious, and many microorganisms have been incriminated. Every veterinarian doing a cattle practice has had to contend with this disease, and has suffered plenty of headaches in attempts to diagnose the particular type of mastitis in a given case and in determining upon the proper treatment.

The importance of mastitis as a cause of epidemics of sore throats and in outbreaks of food poisoning due to streptococci and staphylococci from udder infection has been widely publicized, too widely perhaps. Aside from its importance as a public health problem the cost of mastitis in lowered milk production and loss of valuable animals amply justifies attempts to control it. The war effort necessitating, as it does, greatly increased milk supplies has put dairymen on the alert to combat any disease which threatens to cut their milk production. As a result calls for help in mastitis control have been of alarming frequency in my practice, during the past few months. I have treated more cases of mastitis within six months than during any previous year since I have been in practice. Recent cases of the disease have included every type of mastitis that has ever been described.

The section in which I practice includes a large number of small dairies. There is little official restriction on the sale of milk. The sanitary condition of barns, milkers, milking utensils and other equipment used in its production receives little attention. Heretofore the sale of milk has been chiefly to cheese and butter plants and the producers have been concerned only as to the percentage of butterfat and the pounds of milk. Outbreaks of mastitis are frequent among the herds of this section and the percentage of chronic mastitis in most of them is high.

However, at present more and more of these dairymen are becoming mastitis conscious. The reason being the high price of dairy cows and the fact that milk is selling at a price far above the level of that of recent years. The army camps in our vicinity now take all the milk we produce, and would take more if we had it. When the demand for milk is greater than the supply, the dairyman will combat anything or any disease that cuts his production. Therefore at the present time he is watching all milking cows closely, and at the first symptom of udder trouble the veterinarian is called. News items in various farm journals and radio broadcasts have been a great help in calling the farmer's attention to the importance of controlling mastitis.

Unfortunately some of the leading farm journals have led some dairymen to believe that there is only one kind of mastitis and only one treatment. An article published a few months ago in one farm journal gave some dairymen the idea that all they had to do when a case of mastitis occurred in their herds was to inject a small amount of collodial silver oxide into the udder. There are a few dairymen in my district who still believe this, and no matter what the type of mastitis they inject 10cc or so of silver suspension into the udder. Since streptococcus infection is responsible in a majority of the cases their results on percentage basis is such that they figure they are doing a rather good job.

To the veterinarian mastitis is a complicated disease requiring much study and technical skill for diagnosis; a disease that has many forms and many micro-organisms involved. The farmer from reading reports in his farm papers believes there is but one type of disease, only one organism involved and only one treatment necessary. We must take therefore the time to explain fully to every client having a mastitis problem that there may be different organisms involved and different treatments required.

*Presented at the Midwest Veterinary Conference, Kansas City, Mo., June 30, 1942.

In the earlier days of my practice mastitis was not considered an important problem. The owner would call my attention to a case of "garget" usually when I was on the farm treating some other animal. Once in a while I was called to see a case of acute mastitis, but even in such cases the owner was not particularly concerned except that the life of the animal be saved. But today the increase in the prevalence of mastitis is so great and the importance of maximum milk production is so stressed that this disease has become one of the major sources of income in my practice.

Bovine mastitis may be due to any of a number of bacteria but more than 90% of the cases are due to streptococci. Three different species of this organism are involved. The first in prevalence is *Streptococcus agalactiae*; the second is *Streptococcus uberis*, and the third in importance is *Streptococcus disgalactiae*. These organisms are transmissible readily from cow to cow. The term "hemolytic strep" used in relation to mastitis is often confusing. It refers to hemolytic streptococcus, *Streptococcus epidemicus*, that causes septic sore throat in man. Mastitis in cows is infrequently caused by it owing to the cow being infected by a milker who is infected by the organism or is a carrier of it.

Streptococcus agalactiae is the organism usually found in the diseased udder. Its presence has been reported in some udders that on physical examination appeared to be healthy. Differences of opinion on this point are probably explained by the variation in thoroughness with which clinical examinations are made by different practitioners. The *Str. agalactiae* is capable of existing up to 30 days in the barn outside of the udder and therefore it is unwise to squirt milk from any case of mastitis on the floor. *Str. disgalactiae* and *Str. uberis* are closely related to *Str. agalactiae* and the three are commonly regarded as the mastitis streptococci.

Staphylococci involved in mastitis are of two types. (1) The mild non-clinical type, which is believed to be only a slight udder irritant, but which will finally produce enough scar tissue to lessen milk production, and (2) other strains which cause extreme injury to the udder parenchyma.

The chronic type of staphylococcic mastitis cannot be distinguished clinically, from streptococcic mastitis. The other type of staphylococci usually cause acute mastitis with the loss of the infected quarter, gangrene, or even fatal septicemia. In some cases both streptococci and staphylococci are present in the udder. In occasional outbreaks of mastitis such organisms as *Escherichia coli, Clostridium welchii, Corynebacterium pyogenes, Actinomyces bovis,* and *Brucella abortus* have been isolated.

Classifying mastitis according to its etiology and pathological alterations, is not without its faults; but it may be a more serviceable method than that based on the anatomical structures involved.

1. *Acute Diffused Mastitis.*—An inflammation of the udder, characterized by exudative process in which the exudate may be serous, cellular, fibrinous, or hemorrhagic. The organisms usually involved are staphylococci, *Esch. coli,* and streptococci. This type of mastitis usually occurs in the early stages of lactation.

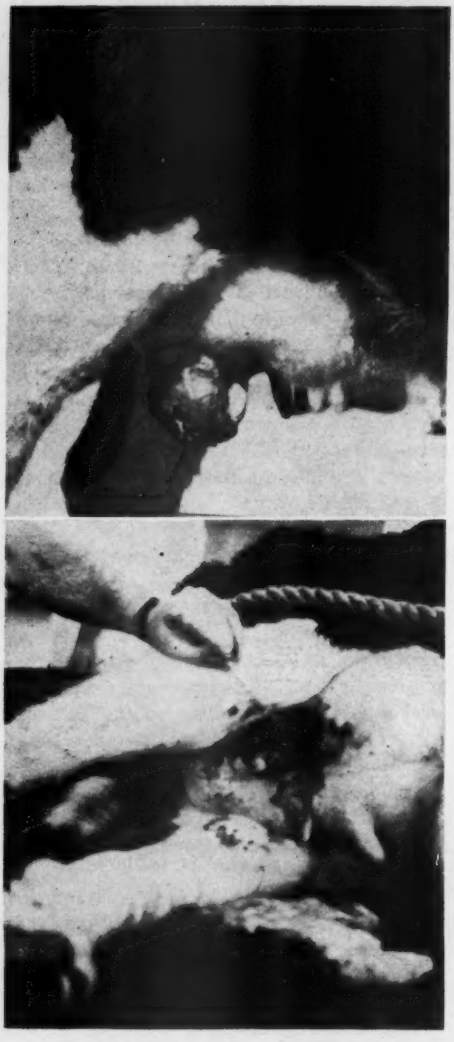
2. *Chronic Diffused Mastitis.*—An inflammation of the udder characterized by atrophy and fibrosis of the gland parenchyma. It usually begins as an exudative or suppurative inflammation of the lactiferous ducts. Secretion is altered (bloody, purulent, serous, or slimy) and often gray in color. In later stages of streptococcic mastitis the milk cistern and milk ducts are filled with a greenish, slimy exudate containing streptococci.

3. *Subacute and Chronic Focal Mastitis.*—The histological changes in the udder in this type of mastitis have for the most part, a focal distribution and are either subacute or chronic in character. The number of these inflammatory foci in an infected udder is variable.

4. *Suppurative Mastitis.*—This type is reported to occur most frequently in dry cows. It is characterized by fibrous capsules. One or more quarters may be affected and the teat canal and cistern may be filled with a greenish, foul-smelling pus.

We have catalogued the groups of or-

ganisms which may cause an outbreak of mastitis in a herd, but we have not mentioned the source of these bacteria. There



One type of staphylococci usually causes acute mastitis, with loss of the infected udder, gangrene (as pictured above) or even fatal septicemia. Sometimes streptococci are also present

are three principal sources of bacteria; (1) badly infected cows, (2) insanitary stables and (3) human carriers of streptococci.

The spread of infection from the diseased cow and contaminated sources is facilitated by bad milking practices.

When cows with diseased udders are milked just before milking healthy animals, the hands of the milkers may carry infection from diseased to healthy udders. This is accomplished by getting infected milk on the ends of the teats, or onto injuries of the udder or teats. This in time leads to entrance of bacteria through the teat canal or through injuries to the mammary glands.

The spread of infection is readily observed when a cow with a normal udder is placed in a group of cows having badly diseased udders. Within from two to three months a fibrosis can be detected in the formerly healthy udder, and in the course of a year the lesions may be marked. In a herd where there is a high percentage of badly diseased quarters and no precautions are taken, many of the normal cows become diseased within a period of six months, and eventually every cow in the herd becomes infected. A group of mastitis free cows may be maintained in a healthy condition over a long period if no badly infected animals are associated with them.

From my observations I am convinced that milkers, both mechanical and human, are the outstanding transmitters of this disease. A few interesting experiments have been carried out by the Florida Agricultural Station indicating that the house fly (*Musca domestica*) may be a vector of bovine mastitis. Mastitis developed in all cattle used in this experiment. This I believe is conclusive evidence that the fly has something to do with the transmission of bovine mastitis. Thus mastitis has the usual background of filth, dirt, disregard of sanitation and unscreened dairy barns.

Additions to a herd by purchase is a loophole through which mastitis may gain entrance into a clean herd. Often 50% of a consignment of dry cows may prove to be infected during the following lactation period. This can be explained in two ways, (1) inflammatory changes, except they be gross, are not easily recognized in the udder of a springing cow, and (2) after an attack

of mastitis the disease is apt to be permanent or recurrent, even though the acute symptoms may disappear. There is the further factor that infected cows are more likely to be put upon the market than are healthy cows.

The second source of mastitis organisms—the insanitary barn—may function in several ways. Injuries predisposing udders to infection may result from faulty stable construction, hooking of the udder, sucking among heifers and stalls that are too short or which are slippery. Bedding can lessen the spread of this disease by lessening exposure to filth, moisture and cold. The use of the milking machine under strict sanitary conditions, and in the hands of men experienced in the use of the mechanism, is a satisfactory way of obtaining the milk with no chance of injuring the udder or of spreading infection. But when improperly cleansed and disinfected, milking machines harbor infection continuously, and infected cows are more apt to go unrecognized when milked by machine than when milked by hand.

One of my clients recently purchased a good herd of 80 cows. It had no history of trouble from mastitis. Included in the purchase was the milking machines and all equipment. The herd and equipment were put in a barn where the conditions were very insanitary. I was called upon to make such tests as I thought necessary. The bromthymol test and a bacteria count of the milk from each cow showed a change in the pH and the presence of excessive numbers of bacteria in the milk of but three animals. The milkers were inexperienced, and had slight knowledge of sanitation and hygiene. Through careless cleansing of milking machine and milkers hands, and filthy bedding 50% of these dairy cows were infected with clinical mastitis in less than four months. This, along with other experiences illustrates, that in the absence of sanitation of milking machines, cleanliness of the udder and teats and isolation of infected cows, bovine mastitis spreads rapidly.

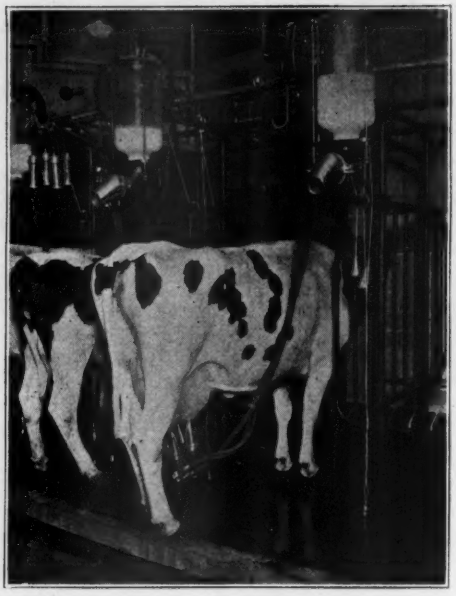
Freedom from infection can be aided materially through sanitary, well-ventilated, and screened cow-barns

Milking on the floor as a means of discarding the milk from a badly diseased quarter cannot be condemned too severely. Carelessness in directing the milk into the pail and thus spilling it on the floor is also to be avoided.

Mechanical injuries which might lead to mastitis can be caused by a milking machine in which the vacuum is set too high. In one herd where a vacuum of 17 pounds was employed, distinct clinical mastitis, with gargety milk developed in a high percentage of the cows. I believe that high vacuum was an important factor in converting a chronic, low grade infection into an acute form of the disease. The infection was apparently of low virulence and the condition of the udders improved very rapidly when the vacuum on the machine was decreased. Rough manipulation in the physical examination, or stripping with the thumb and forefinger, may help to develop mastitis, or to extend a small foci of infection already present. Wet milking is an offense against the quality of the milk and the udder, and any milker who persists in it should be barred from the stable. Milking tubes and dilaters are a menace in the hands of the average milker; likely to

result in introducing new infections or aggravating those already present.

Running calves with the cow to keep the quarters milked out is a common practice in acute mastitis. In certain sections this



In this type of milking machine, the milk is carried directly from the udder to the glass jar above without exposure to the air or other contamination

is the usual method of rearing calves. Two or three calves are usually turned in with the cow. Observations of the udders of nurse cows suggests that the practice predisposes to inflammation of the udder. It has been noted that some cows with normal udders develop induration after being nursed by two or three calves for two or three months. Calves sucking one another after drinking milk may develop an infection which is not recognized until the heifer comes into lactation. Washing the udder in a cold stable and not drying it predisposes to mastitis, and repeated use of a wiping cloth without sterilizing it exposes the udder to the possibility of infection carried from cow to cow.

The period of lactation is another important factor in the development of this disease. The beginning and the end of lactation places a heavy strain on the udder and at these times a slumbering infection is apt to become active. At these periods the udder needs special care. A requirement often neglected, in "drying off" cows is to substitute hay for grain. After regular milking has been discontinued the udder should be watched closely. Latent or mild mastitis may be aggravated to an acute attack. In such cases it is better to continue milking the quarter and not give it a period of rest. Injury may result from insufficient removal of milk from the udder directly after freshening. When milking is left to the calf, excessive engorgement of the already hypersensitive gland may cause an acute inflamation. In my practice, mastitis is more prevalent in the larger herds because the animals in such herds receive less individual attention than in small herds. However, this does not obtain in more intensive dairy districts. There milk production is the principal business of the farmer, and the large are better cared for than the small herds.

It is common observation that this disease is more prevalent among the older cows in a herd. I see very few old cows that have been heavy producers that are entirely free from udder fibrosis. In badly infected herds, where the young and old are together, the mastitis rate is lowest in the younger group. The reason is probably because they have had fewest opportunities to become infected. Infection enters the udder less readily if the teat meatus is tightly constructed. If the meatus is so relaxed that milk leaks when the cow "gives down," sooner or later infection is almost certain to enter the gland and cause infectious mastitis.

The practitioner who is engaged in cattle practice to any considerable degree should be able to determine the type of infection in this disease. As has been said most cases of mastitis are a result of streptococcus infection. There are several methods by which the organism involved in a given case may be determined in a considerable percentage of the cases without the help of a laboratory. The first test and a most important step in the preliminary examination is the use of a strip cup. This will

detect gross abnormalities in the milk, flakes, clots, changes in color and consistency. The strip cup is particularly useful in detecting first stages of an acute inflammation. A second step in the examination is the thybromol test. It is based on the change in the pH of the milk. The details of the test are:

Discard the first few streams of milk, then collect 5cc in a clean test tube. Examine the sample for color and consistency. Then add 0.5cc of thybromol indicator solution. Normal milk will become slightly yellowish green. A positive reaction is indicated by a definite green color. The intensity of the green varies with the alkalinity of the sample.

If the infection is due to staphlococci the test is less than 25% accurate; if due to streptococci it is about 50% accurate.

A third method of detecting mastitis and one fairly accurate for streptococci infection is the Hotis test. This test is simple and is readily carried out by the practitioner. The procedure is as follows:

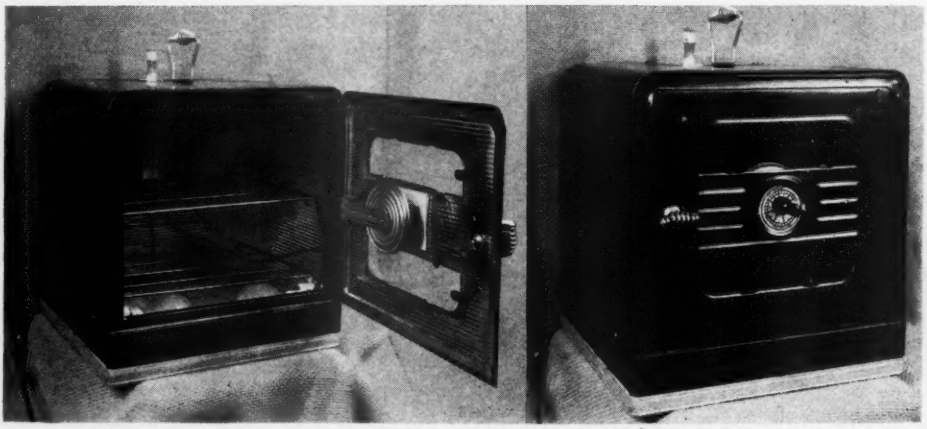
To 9.5cc of fresh milk, aseptically collected from the suspected quarter, add 0.5cc of 0.5% aqueous bromcresolpurple solution. Incubate at 37.5% C. (98.6° F.) for 24 hours. If the *Str. agalactiae* is present a thick yellow deposit forms in the bottom of the tube or small yellow beads of growth and flakes adhere to the sides of the tube.

Small incubators sufficient for the purpose can be made at little cost, or may be purchased for a few dollars from supply houses. The solution for the Hotis test can be purchased from any commercial laboratory.

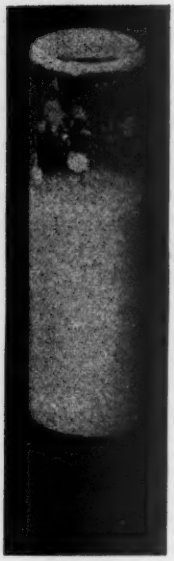
A fourth and by far the most accurate and dependable laboratory test for the detection of mastitis is the bacteriological examination. By this procedure it is possible to determine definitely the type or types of bacteria involved. It is strictly a laboratory procedure, and requires much equipment, good technique and ample experience. It is not a test for the practitioner.

A simple but revealing bacteriological examination, which is feasible for the practitioner who has the time, is made of properly stained smears prepared from 12-hour incubated samples of milk. It is not possible to determine by this examination whether the streptococci, if found, are pathologically significant, therefore the samples must be properly collected to avoid contamination from the non-pathogenic. This examination will enable the veterinarian to detect the normal cows and thus by exclusion locate the infected animals.

All the foregoing tests are merely supplemental to the physical examination of the udder, and are to be interpreted in the light of the findings on physical examination. It is very important to know whether the infection is recent or whether marked fibrosis has taken place. Furthermore, for accurate prognosis, it is vital to know the



Two views of a bacteriological incubator, constructed at small expense by C. S. Bryan and L. F. Jennings, Michigan Agricultural Experiment Station



Incubation of milk samples for 24 hours at 37.5°C. (98.6°F.) is a simple and useful method of testing milk from a suspected quarter. If the *Str. agalactiae* is present, a thick yellow deposit forms in the bottom of the tube, or small yellow beads of growth and flakes adhere to its sides

exact condition of the mammary tissue in order to interpret the results of the other tests. Routinely when called upon to examine a herd for the existence of mastitis, I used the strip cup first to detect abnormalities, if any, in the milk. Second, samples are collected for the thybromol test and also for the Hotis test, and in some cases, for the bacteriological test. The quarters are then all stripped clean and a physical examination of the udder is made.

After completing these four steps and obtaining such information as the tests reveal I feel that I am able to make an accurate diagnosis and to determine the treatment required. Only the physical examination reveals whether the cow should be slaughtered immediately. In general I segregate all infected animals immediately, employ strict sanitation where the milking is done, screen the dairy barn completely and insist upon cleanliness of milkers and milking utensils.

For the treatment of steptococci mastitis the selection of the proper sulfa compounds is an important point of departure. In most cases of acute mastitis due to the staphylococci, *E. coli, Coryn. pyogenes* sulfathiazole appears to be more beneficial than sulfanilamide. I have used sulfanilamide in the treatment of various cases of streptococci infection for several years, with fair results, particularly in acute diffuse mastitis. After administering sulfanilamide the acute attack often subsides and a normal appetite returns but examination of the milk a few days later reveals streptococci in the infected quarter. Therefore, I believe sulfanilamide has a place in mastitis treatment. Before I used it in these acute cases I lost some of them. It certainly saves the life of the animal in the acute form of streptococcic mastitis and gives us an opportunity to employ other treatment later to complete the cure where necessary.

I have used the infusion method of treating mastitis with satisfactory results. At present I am using a modification of Steck's treatment—the infusion of a 1 to 1500 dilution of acriflavine in 20% dextrose solution. Steck determined that the pathogenic organisms are confined to the lower, most readily accessible part of the quarter. The infusion may thus be limited to the cistern of each quarter. The infusion of a large volume of fluid is unnecessary and, especially if given under pressure, results in avoidable irritation. In lactating cows inject not more than 300 to 400cc of the solution into the infected quarter by gravity; then milk it out within five minutes. I have obtained a high percentage of cures following this treatment. In hot, swollen, and painful udders the infusion may be allowed to remain in the udders for ten minutes. When dry udders are treated we strip the quarter about twice a day for three or four days. I have observed very little reaction from the infusion of acriflavine and dextrose while the injection is being made. In some cases the animals kick a few times in the other cases they show a little uneasiness by moving about or by slightly labored breathing but this passes in from 5 to 15 minutes. No late reactions have been noted.

The infusion of colloidal silver oxide has received a large amount of publicity. Personally I have used it less than the acriflavine having treated about 80 cases with it. The results have been fair. From about 15% of the udders infused with silver oxide a few sterptococci were isolated later. This

preparation is relatively nonirritant to the tissues, yet is bactericidal. It consists of 5% of tiny flakes of silver oxide suspended in mineral oil. The particles of silver are so small that they permeate the skin and tissues readily and speedily. I inject 10cc into each infected quarter of dry cows and leave it there. In lactating cows I inject 5cc and in 24 hours milk it out, and then inject 10cc at the end of a second 24-hour period and milk it out. Another 10cc is injected immediately and allowed to remain. The three injections has so far eliminated *Str. agalactiae* from large percentage of the udders of the lactating cows on which I have used it.

D. A. Sanders, Gainsville, Florida, reports good results from infusion of udder with a 1 to 1000 to 1 to 1500 solution of resublimed iodine crystals in liquid petrolatum. From 100 to 300cc are injected into the infected quarter. He has reported no permanent damage to the udder tissue but that the injection caused slight irritation which diminished the milk flow temporarily. The flow returned to normal in 10 days. I have not used this treatment.

A remarkable new treatment for mastitis comprises the infusion of gramicidin. This is a substance, isolated by Duboe from cultures of aerobic sporulating bacteria from the soil. It is soluble in alcohol but insoluble in water. It is stated to be more lethal to the *Str. agalactiae* than anything heretofore employed in udder fusion. Very recently I have obtained a quantity of gramicidin but as yet have had no opportunity to use it. C. S. Bryan of the Michigan Experiment Station summarizes his results with it as follows:

"One hundred and fifty-seven cows with streptococcic mastitis were treated by infusing each quarter with 150mg of tyrothricin. Of these 142 or 90% became free from the streptococcic infection of the udder.

"Three cows did not drop in milk production upon treatment while 64 produced from 1 to 50% less milk, and only six cows of 73 dropped between 50 and 90% in production. All returned to approximately normal milk flow within 21 days.

"The period of abnormal milk production after treatment of 73 cows was less than five days for 57 cows and more than five but less than 14 days for the remaining 16 cows.

"The standard plate bacteria count of milk produced by 10 cows before treatment and upon recovery after treatment yielded reductions in bacteria count varying from 30 to 99%."

In acute mastitis, sometimes two or three calves are allowed to run with a fresh cow to keep the quarters milked

Autogenous bacterins are a valuable agent in the treatment of mastitis. In so-called mixed infection mastitis, autogenous bacterin is the answer. I have treated five herds this year with autogenous bacterins with satisfactory results. Milk is taken from all four quarters of each cow. From this composite collection a bacterin is made. As a treatment it is not perfect, but it is good enough to be satisfying. Under the name of Lactovaccine Doctor Bryan[1] gives the method of preparing and using this vaccine as follows:

A herd-autogenous or an animal-autogenous vaccine, depending upon the number of cases to be treated, was prepared by aseptically milking some of the milk containing streptococci from the infected quarters into a sterile glass bottle. The amount of vaccine prepared each week depended upon the number of cases that were treated. To the streptococcus-infected milk was added a 1 per cent (1:100 dilution) aqueous solution of either gentian violet or brilliant green, so that the final dilution of the dye in the milk was 1:10,000. Adding dye (1 per cent solution) at the rate of 0.1 to 10cc of infected milk gives the proper dilution of 1:10,000. The dye and milk were mixed thoroughly and allowed to remain in contact 30 minutes, with occasional shaking before this lacto-vaccine was used for injection.

[1] Bryan, C. S., 1933. Studies on vaccine in infective mastitis; *Jnl. A.V.M.A.*, 36:3, pp. 390-399.

Isolation of Western Equine Encephalomyelitis and Hog-cholera Viruses from Supposedly Hog-cholera Immune Swine

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DURING the course of a survey in an attempt to learn something of the incidence of Aujeszky's disease in swine, hog-cholera virus, existing under what appeared to be peculiar circumstances, and western equine encephalomyelitis virus were found in the tissues of swine. These findings are reported herein.

In the survey of Aujeszky's disease, to be reported fully at a later date, an appreciable number of hogs have been examined for this disease. These examinations have been made of the tissues coming to a laboratory for routine diagnosis. Except in two cases, the results have been negative. In one instance a farmer lost 105 of 176 baby pigs from Aujeszky's disease. In the second instance, one serial of commercial hog-cholera virus was found to have caused paralysis in small pigs when it was used in immunizing against hog-cholera. The inoculation of experimental animals with nerve tissue from a number of these small, paralyzed pigs revealed a virus identical to that which causes Aujeszky's disease. It is concluded that one or more of the pigs from which the hog-cholera virus was prepared were infected, inapparently, with this disease.

Hog-cholera virus and the equine encephalomyelitis virus were isolated during the examination of two pigs, both from a herd of 56 young animals. The hogs in this herd had been vaccinated with hog-cholera virus and antiserum June 4, 1941 at an average weight of about 35 pounds. The entire lot remained healthy until July 10 when the first animal sickened and July 14 when the second became ill. There was no more sickness in the herd thereafter. The ailing pigs were killed and examined in the laboratory on the dates indicated above. Both pigs were found to be suffering from an icterus of hogs which is seen usually in coal tar pitch poisoning as reported by Van Es,[1] and by Graham, Hester, and Henderson.[2] There is no reason to suspect that this particular type of icterus is caused by infection, although it is possible that other poisons may produce a like pathological picture. Coal tar pitch poisoning usually causes death within a few hours after symptoms are observed. Neither pig showed gross evidence of any other disease.

Aujeszky's disease is usually a sub-clinical ailment in swine; discovered only when the infection in cattle, in which it is practically always fatal, is discovered on the same premises. Swine are thus regarded as the natural reservoir of the infection. The authors demonstrate that in young animals it may be a serious affection of swine.

■ Cultures from the tissues of the first pig, No. 1604, showed *Salmonella choleraesuis*. A suspension of liver and spleen from this first pig caused no disease symptoms when inoculated subcutaneously or intraperitoneally into rabbits and cholera immune hogs, but when inoculated subdurally into rabbits and guinea pigs it caused death with a streptococcus infection in 24 to 48 hours. A brain suspension from one of these inoculated rabbits, dead in 24 hours, and a liver and spleen suspension from pig No. 1604 were inoculated into cholera susceptible pigs Nos. 1624 and 1625 respectively. Both pigs sick-

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[1] Van Es, L., 1941. University of Nebraska, Lincoln, Nebraska, Personal communication.

[2] Graham, R., H. R. Hester, and J. A. Henderson, 1940. *Jnl. A. V. M. A.* 96 (Feb.); pp. 135-140.

ened in six days with symptoms of hog-cholera. Uninoculated cholera susceptible control pigs remained healthy. Upon appearance of the symptoms, pig No. 1624 was given 50cc of hog-cholera antiserum and it recovered. When challenged with cholera virus 42 days after receiving the immune serum this pig was found to be immune. The untreated pig No. 1625, died, and, upon postmortem examination lesions of cholera were found. Thus, pig No. 1604 was harboring hog-cholera virus 36 days after having been vaccinated against the disease, yet the evidence indicates it was "immune" and the virus was causing no apparent disease. Results also showed that the virus survived in the brain of the rabbit for 24 hours. Finally, as an additional test to determine whether any other infection was present, a brain suspension from pig No. 1625 was inoculated subdurally into a rabbit. This animal remained healthy.

Examination of the second original pig No. 1614 also showed the hog-cholera virus to be present. When susceptible pigs were injected subcutaneously with tissue suspensions from this pig they died of hog-cholera promptly, whereas immune pigs remained healthy. As an example, susceptible pig No. 1626 sickened with symptoms of cholera five days after injection. It was killed on the seventh day and showed lesions of cholera. Susceptible pig No. 1642 remained healthy following injection of 40cc of hog-cholera anti-serum plus 5cc of 20% brain suspension from pig No. 1614. Uninoculated control pigs also remained healthy.

■ This second pig, No. 1614, is of further interest because still another totally different virus was isolated from it. When a 10% suspension of liver and spleen from the second pig, No. 1614, was injected subdurally into three healthy guinea pigs, they all sickened on the fourth day thereafter and were killed on the fifth day for culture and to harvest tissues for further animal inoculations. When a like suspension of brain tissue from hog No. 1614 was injected similarly into three other guinea pigs, they remained healthy showing that the disease producing agent was not present in the brain of this hog.

The infectious agent from the first group of guinea pigs was then carried in series through guinea pigs by brain to brain inoculations and is still being so propagated. Intraperitoneal inoculation in guinea pigs was uncertain and hence unsatisfactory. In this regard it was observed that guinea pigs which withstood an intraperitoneal inoculation were usually immune to a later intracranial inoculation even though they showed little or no reaction from the first injection. It was also found that guinea pigs that had recovered from the disease produced by either route of exposure were usually solidly immune to a second exposure either intracranial or otherwise. Calves and rabbits remained apparently healthy following subdural inoculation. Berkefeld N filters were employed in preparing filtrates. Such filtrates reproduced the disease in susceptible guinea pigs exactly as did unfiltered material, although the onset of symptoms was sometimes delayed for a day or so. When such filtrates were first inoculated into the allantoic sac of developing chick embryos, results were negative. When the same filtrates were injected into the yolk sac of similar developing chick embryos the embryos always died within 24 hours. Once the infectious agent had been established in developing chick embryos by the yolk sac route, it was then easily propagated in chick embryos by the allantoic sac inoculation method.

Guinea pigs that had recovered from the infection did not sicken when injected with a challenge dose of the virus. They were then inoculated subdurally with a strain of known western equine encephalomyelitis virus* and again did not sicken, whereas controls sickened promptly. In a similar manner the

* Kindly furnished by Dr. H. J. Shore of the Ft. Dodge Serum Co., Ft. Dodge, Iowa.

virus isolated from the pig was also compared to another strain** recently typed from horses and found to be western equine encephalomyelitis. Again the "pig virus" proved to be immunologically identical.

■ The finding of the western equine encephalomyelitis virus in the tissues of a pig obviously raised many questions. If hogs can be so infected, do they show apparent disease? How often are hogs apparently or inapparently infected? Do hogs act as a reservoir for the equine virus? Attempts were made to furnish at least partial answers to these and other questions. Two hogs were fed the brains of four guinea pigs that were infected with the pig strain of equine encephalomyelitis. These hogs were also fed several pints of the same strain of virus propagated in chick embryos. They showed no evidence of disease during a period of observation extending over several weeks. Hogs weighing 60 to 200 pounds were injected subdurally, subcutaneously, or intravenously and in some instances were also fed chick embryo propagated virus. Following such exposure, attempts were made to re-isolate the virus from their tissues after varying lengths of time. Since these examinations were negative, it does not seem necessary to present the detailed data. To illustrate, however, the results of findings on two pigs can be mentioned. A sow pig weighing 85 pounds was injected subdurally with 1cc of a 10% suspension of brain from an infected guinea pig. Five days later the sow pig was killed and its brain removed. Two bacteria-free filtrates were made of brain suspensions from this pig. Each filtrate was inoculated subdurally into three guinea pigs and into 12 developing chick embryos. No evidence of virus was found in either instance. A boar pig was injected subdurally with a brain suspension from an infected guinea pig. This hog was killed 48 days later and an effort made to find the virus in the hepatic lymph nodes, liver, spleen, and brain. The results were negative. Since exposed hogs did not sicken and the virus was not re-isolated from such animals, attempt was made to find neutralizing antibodies in the sera of swine with a known exposure.

The list of mammals and birds that are known to be susceptible to infection with encephalomyelitis virus by artificial inoculation is already large but few have been found infected naturally. The authors have added swine to the latter list. The hazard of this virus for man and the close association of swine with both horses and mankind place the authors' discovery among the potentially important in connection with this disease.

■ Two hogs weighing 175 pounds each were employed. Both were bled before exposure to the western equine encephalomyelitis virus. Hog No. 6465 was injected intravenously with 0.3cc of chick embryo propagated virus and fed for several days with large amounts of chick embryo and guinea pig propagated virus. Twenty days later blood was again obtained. Guinea pig brain virus was made up in a range of dilutions from 1:40,000 to 1:10,000,000. When these dilutions were mixed with equal parts of sera and allowed to stand overnight, it was found that serum of hog No. 6465, after exposure, protected all the guinea pigs employed in all dilutions, whereas the serum obtained before exposure did not protect the guinea pigs receiving 0.05cc of "neutralized" virus in 1:40,000 dilution. Similarly, hog No. 6466 was fed large amounts of guinea pig and chick embryo propagated virus for about 10 days and was bled just before exposure and 29 days after the first feeding. Western equine virus was mixed in equal parts with the sera, taken both before and after exposure, in dilutions ranging from 1:20,000 to 1:1,000,000. Again, the serum after exposure protected all guinea pigs in all dilutions. One of two guinea pigs in the 1:20,000 and one of the two guinea pigs in the 1:40,000 virus dilutions lots died when injected with the virus "neutralized" with serum taken from the hog before exposure.

** Received from Dr. H. W. Schoening, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Finally, it was thought that age might be a factor in the susceptibility of pigs for equine encephalomyelitis as is the case in Aujeszky's disease. Hence, two 3 day old pigs were each injected subdurally with 0.2cc of chick embryo propagated "pig strain" equine encephalomyelitis virus. Tremor was soon evident in both animals. The symptom persisted for two or three days in one pig after which it became normal. When killed eight days later, tests failed to show virus to be present in its tissues. The second pig became rapidly worse so that it was near death 38 hours later when it was killed. Histopathological study of its brain showed early stages of extensive meningitis. Assay of its brain revealed a high concentration of equine encephalomyelitis virus. Tests of its liver and spleen were negative.

Discussion

Following anti-hog-cholera treatment, it is not unusual to find a few pigs that are still susceptible to hog-cholera. If exposed, such pigs contract the disease as readily as non-vaccinated animals. However, the two pigs reported above showed no symptoms or lesions that could be attributed with certainty to hog-cholera. It is also unusual that the virus should involve only the two pigs that were affected with a disease resembling the coal tar pitch poisoning. The finding of hog-cholera virus in these pigs at least raises the question as to whether vaccinated pigs are always as free of hog-cholera virus after 21 days as heretofore supposed.

No "carriers" of hog cholera virus have ever been recognized although the epizootiology of the disease suggests that they occur, and they have been sought in divers mammals. The authors' experience suggests that searchers may have looked too far afield.

At the time the strain of western equine encephalomyelitis virus was isolated from the above pig, no work was in progress with this disease. No inoculations of guinea pigs with equine encephalomyelitis had been made for several months. The room where the inoculated pigs were kept had had no infected animals in it for several months. Also, no accidental infections with this or other viruses have been experienced over the past several years. When one considers that all three of the first injected guinea pigs sickened and that there was little or no chance of accidental contamination, it seems certain that the virus of western equine encephalomyelitis was present in the tissues of the one hog under consideration. In other words, this is a case of natural infection in which the virus was isolated from the tissues. The virus was present in a pooled sample of liver and spleen but not in the brain. In the small pig that died following artificial exposure the virus was present in the brain but not in the liver or spleen. Hammon, *et al*[3] report that one pig of four tested showed neutralizing antibodies to equine encephalitis in its serum. Mitchell and Walker[4] and Giltner and Shahan[5] have shown that swine are refractory to infection with western equine encephalomyelitis virus.

Summary

1. Hog-cholera virus was isolated under what appears to be unusual circumstances.

2. The virus of western equine encephalomyelitis was isolated from a pig.

3. That hogs are resistant to western equine encephalitis is indicated by failure to produce apparent symptoms in artificially exposed hogs, except in one baby pig and by failure, except in one instance, to isolate the virus from the tissues of exposed individuals.

4. That hogs may be infected, under certain circumstances, with western equine encephalomyelitis is shown by the isolation of the virus from a pig, death and re-isolation of the virus from one artificially exposed baby pig and an increased neutralizing effect of the sera of pigs which had been exposed to the virus.

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[3] Hammon, W. M., J. A. Gray, F. C. Evans, E. M. Izumi, and H. W. Lundy, 1941. *Sci.* 94 (Sept. 26); No. 2439, pp. 305-307.

[4] Mitchell, C. A., and R. V. L. Walker, 1941. *Canad. Jnl. Comp. Med.* and *Vet. Sci.* 5; pp. 314-319.

[5] Giltner, L. T., and M. S. Shahan, 1936. *Jnl. A. V. M. A.*, 88; p. 363.

Sulfathiazole for Colds in Chickens

By VICTOR HEIMAN*, Ph.D.,
Waverly, N. Y.

AN EPIZOOTIC of colds among broilers or layers nearly always means slower growth and/or lower egg production. Some death losses may result also but as a rule the greatest damage is from loss of appetite, unthrifty condition, stunted growth or reduced production.

The usual symptoms are watery inflamed eyes, and nostrils clogged with mucous.

Fig. 1. The swollen watery eye that was a frequent symptom of a cold

Sneezing, coughing and labored breathing are common. In some cases the eyes and face become swollen. In chronic infections of mature birds there is a characteristic disagreeable odor by which the disease may be easily identified. In the white feathered breeds, birds with colds have dirty feathers on the back and shoulders where they have wiped their nostrils.

Treatments usually recommended are intended mainly to keep the birds warm and comfortable, and to relieve, insofar as possible, the congestion of the mucous membrane of the nasal passages and the respiratory tract, and to prevent the spread of infection.

In 1941 Delaplane and Stuart, of the Rhode Island Agricultural Experiment Station, published a report of their experiments on treating colds with sulfathiazole. They showed that sulfathiazole, one of the members of the remarkable sulfa drug family, was effective in preventing and perhaps curing colds brought on by laboratory produced infection. These scientists found that the drug could be given in the feed, that the mature birds ate the feed containing sulfathiazole readily, and apparently there were no poisonous after effects. The drug was effective only as long as it was being fed. Its effects were not cumulative.

At this laboratory it has been our aim to find whether results similar to those produced in the laboratory, could be obtained in a natural outbreak of the disease under practical conditions; whether younger birds would have the same response as mature birds; and how the drug feeding would affect growth.

An experiment was therefore undertaken to answer these questions. A farm was selected for this work which had been having outbreaks of colds in young chicks raised entirely in batteries for broilers.

Two hundred and forty four-week-old Rock X Red cross-bred chicks were selected from a group, many of which already had colds in varying degrees of severity. Only those birds which showed no visible symptoms of colds were selected though all had been exposed to the infection and probably many were developing symptoms which were not observable at the time the experiment started.

The chicks were weighed and wing-banded at the start. They were divided into six groups of 40 chicks each. Groups No. 1, 3 and 5 received an all-mash commercial broiler ration without any supplement. Groups No. 2, 4 and 6 received the same ration but with ⅛gm of sulfathiazole per ounce of feed (this is about seven ounces of the drug in 100 pounds of feed).

In this experiment the arrangement of the battery compartments was important because the intention was to place all chicks

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in the same environment and to provide a point of contact between the treated and untreated pens so that any colds might be readily transmitted from one group of chicks to the other. Therefore, groups No. 1 and 2 were placed at the same height in the growing battery being separated by wire partitions, but both groups drank from the same water trough. The identical arrangement was provided for groups No. 2 and 3, also groups No. 4 and 5. The batteries housing these experimental chicks were in a large room having a number of other batteries holding chicks of various ages, many of which had colds of varying severity. The arrangement was ideal for an experiment of this kind because a natural outbreak of the disease was almost a certainty.

All chicks were individually weighed after the experiment had been in progress two weeks and observations of each were made for the presence and extent of cold symptoms. Further weighings and observations were made as indicated in the results.

The average results in terms of the occurrence of colds are shown in the chart (Fig. 3). Here the records of the three treated groups were combined as were those of the untreated groups, to indicate average results. There were some colds in all groups but as long as the ration containing sulfathiazole was being fed, not more than 20% of the chicks receiving it showed symptoms of colds in any degree. Where this drug was not included in the feed, the incidence of the disease was much higher —90% of the chicks having developed symptoms. This is a highly significant difference.

Fig. 2. This is the arrangement of batteries in the room where this experiment was carried on

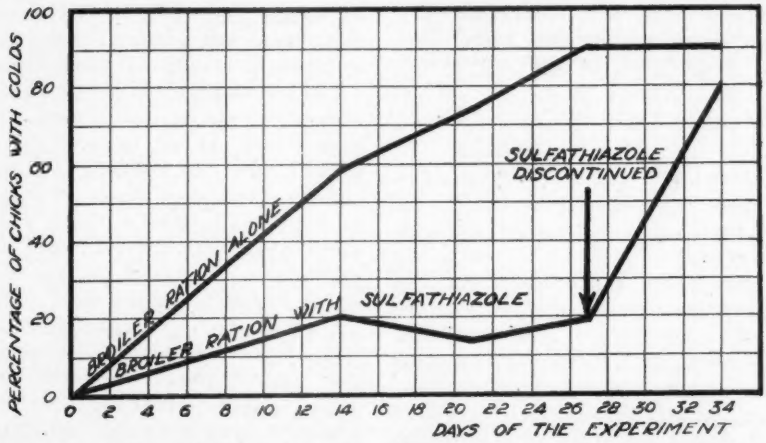

Fig. 3. The feed containing sulfathiazole definitely reduced the number of colds in chicks. When the drug was taken out of the feed, there was a large increase in the percentage of chicks with colds

On the 27th day of the experiment, the sulfathiazole supplement was discontinued, and all groups received the same commercial broiler mash. In a week the symptoms of colds appeared in 80% of the chicks which had previously received sulfathiazole.

This striking result is indicative of sev-

eral things; (1) all chicks were exposed to colds throughout the experiment; (2) sulfathiazole in the amounts fed in the experiments had a definite protective effect against colds in a large proportion of the chicks; and (3) the drug does not make chicks permanently immune to colds and is a protective aid only while it is present in the blood stream.

As for the effect of feeding sulfathiazole on growth this result may be seen in Table I. In every case where the drug was administered in the feed, the average weight

TABLE I. THE EFFECT OF SULFATHIAZOLE IN THE FEED ON THE GROWTH OF CHICKS EXPOSED TO COLDS

Lot no.	Sulfathiazole in ration	Av. body weight at start of exp. ounces	Av. body wt. after 27 days ounces	Average gain in body wt. ounces
1	no	8.6	20.0	11.4
2	yes	9.0	23.1	14.1
3	no	8.9	20.5	11.6
4	yes	8.7	23.0	14.3
5	no	8.7	23.1	14.4
6	yes	8.7	24.9	16.2

was greater at the end of the experiment. This greater average body weight was probably due to the fact that the chicks receiving the drug had fewer colds and therefore less interference with their growth.

There is much work to be done on this problem before definite recommendations can be given on the use of sulfathiazole for the treatment and prevention of colds in chicks. Here is evidence, however, sufficiently convincing to encourage the hope that when further facts are added to our present knowledge, a practical method of dealing with colds in chickens will be available. Besides the research still to be done, it will be necessary to take into consideration the cost and availability of the drug. All these and other related problems have yet to be solved. Sulfathiazole may be the answer to the question, "My chickens have colds, what shall I do for them?"

Acknowledgement

*We wish to express our thanks to Dr. C. A. Bottorff, poultry pathologist of Lederle Laboratories, for supplying the sulfathiazole and for his generous assistance in conducting this experiment. We appreciate also the cooperation and help given by Mr. Charles Bower of the Dugan Brothers Greenbrook Poultry Farm, where this experiment was conducted.

Tuberculosis Eradication a Professional Achievement

By 1918 the losses from tuberculosis were appalling; 4% of the cattle and about one hog in five which came to our great packing houses were infected. City health authorities were threatening the dairy industry with exclusion of its products from the cities. The federal Bureau of Animal Industry, in cooperation with the state livestock sanitary authorities, undertook the eradication of this widespread and enormously costly disease of animals. Profiting by its painful experience in tick eradication and in hog cholera control, the government set up the tuberculosis eradication project as a strictly veterinary project, the direction and conduct of which was begun and maintained throughout exclusively in professional hands, against enormous pressure from political forces in many of the states. The project was undertaken 23 years ago. Today, no state in the whole United States has more than 0.5% of tuberculosis among its cattle. Much remains to be done to eliminate from our cattle the last remnants of the disease, but the loss, except for minor instances, is mainly over; the danger to the public health past. Nearly a quarter billion tuberculin tests have been made by veterinarians and almost four million reacting animals have been removed from the cattle herds of the United States. Every herd has been tested one or more times. Throughout the whole project, from the man in Washington who planned it to the man who tested the cattle in the dairy barn or on the range, the work has been in the hands of men who were educated to handle disease—men who knew the job they were expected to do. The condemned animals were appraised by veterinarians and paid for on their certification. The premises were disinfected under the supervision of veterinarians and the carcasses of the slaughtered animals were inspected by veterinarians. It was a grand job, and not a breath of scandal has attached to it from the beginning to the present moment. However, that record is not surprising; it is expected from any professional service.

★ ★ ★ BUY WAR SAVINGS STAMPS AND BONDS

Autobiography of a Veterinary Practitioner

By E. T. BAKER, Moscow, Idaho

X

BEGINNING PRACTICE

IN JULY, 1913, I began practice in earnest. I was just 30, with four years of diversified experience, and possessed good health. I do not think young men realize what a good field or a poor field means to them. A good location is where the people have enough livestock to support a veterinarian, and are accustomed to summoning expert help. Then, again, if the practitioners before him have been competent veterinarians and good citizens, the task is just that much easier. To locate in a community where the preceding practitioners have been unscrupulous drunkards or immoral scamps requires a good many years of "building up."

I had no car at that time, but hired one from the livery stable. They had an old auto—a Chalmers, I think, that had to be cranked—and it was an all day's job to change tires. It wheezed along the dirt roads at nearly 20 miles an hour and, considering the care it received, always performed nobly for me. In bad weather, or whenever it rained, the roads were impassable for the car, so a team and buggy was used. As I did all the veterinary work for the livery stable, our bills just about coincided, for I always presented my bill last.

The owners of this livery stable were two old brothers who had come west as boys in a covered wagon. They had homesteaded in the early days, and both were well off. They were lovers of good horses, and spent most of their spare time "cussing" the new fangled autos and trucks. They both passed on some 20 years ago, the last of a vanishing generation.

For $12 a month, we had rented a very comfortable house—one that, in this section, would bring around $35 today. Our first experience in renting was our last; as it turned out, we were fortunate in having a grasping landlord. When we moved into this place, it looked like a typical "renter house." The first thing we did was to purchase a lawn mower and some hose, and spend three or four dollars a month in watering the lawn and keeping it in good condition. For this expense and trouble, our landlord dropped over one beautiful afternoon and informed us the rent would be $18 a month from then on, as he could get that much for it now. We were so indignant, we started out to look for a house or vacant lot, and decided on our present location, where we built in 1916. However, we rented for several more years, and saved up enough to pay for our new home when we moved into it. The total cost, including pavement and various improvements, was around $6,500.

Perhaps this experience was the best thing that could have happened to us, for saving and sacrificing was a pleasure whenever we thought of how our landlord had charged us extra for improving his property. We have never been in debt since we were married, as we never purchase things until we can pay for them. By being patient, and buying a few essentials each year and paying for them, it is remarkable how soon one can have a comfortable home. Best of all, when one is out of debt, and hard

eral things; (1) all chicks were exposed to colds throughout the experiment; (2) sulfathiazole in the amounts fed in the experiments had a definite protective effect against colds in a large proportion of the chicks; and (3) the drug does not make chicks permanently immune to colds and is a protective aid only while it is present in the blood stream.

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TABLE I. THE EFFECT OF SULFATHIAZOLE IN THE FEED ON THE GROWTH OF CHICKS EXPOSED TO COLDS

Lot no.	Sulfathiazole in ration	Av. body weight at start of exp. ounces	Av. body wt. after 27 days ounces	Average gain in body wt. ounces
1	no	8.6	20.0	11.4
2	yes	9.0	23.1	14.1
3	no	8.9	20.5	11.6
4	yes	8.7	23.0	14.3
5	no	8.7	23.1	14.4
6	yes	8.7	24.9	16.2

was greater at the end of the experiment. This greater average body weight was probably due to the fact that the chicks receiving the drug had fewer colds and therefore less interference with their growth.

There is much work to be done on this problem before definite recommendations can be given on the use of sulfathiazole for the treatment and prevention of colds in chicks. Here is evidence, however, sufficiently convincing to encourage the hope that when further facts are added to our present knowledge, a practical method of dealing with colds in chickens will be available. Besides the research still to be done, it will be necessary to take into consideration the cost and availability of the drug. All these and other related problems have yet to be solved. Sulfathiazole may be the answer to the question, "My chickens have colds, what shall I do for them?"

Acknowledgement

*We wish to express our thanks to Dr. C. A. Bottorff, poultry pathologist of Lederle Laboratories, for supplying the sulfathiazole and for his generous assistance in conducting this experiment. We appreciate also the cooperation and help given by Mr. Charles Bower of the Dugan Brothers Greenbrook Poultry Farm, where this experiment was conducted.

Tuberculosis Eradication a Professional Achievement

By 1918 the losses from tuberculosis were appalling; 4% of the cattle and about one hog in five which came to our great packing houses were infected. City health authorities were threatening the dairy industry with exclusion of its products from the cities. The federal Bureau of Animal Industry, in cooperation with the state livestock sanitary authorities, undertook the eradication of this widespread and enormously costly disease of animals. Profiting by its painful experience in tick eradication and in hog cholera control, the government set up the tuberculosis eradication project as a strictly veterinary project, the direction and conduct of which was begun and maintained throughout exclusively in professional hands, against enormous pressure from political forces in many of the states. The project was undertaken 23 years ago. Today, no state in the whole United States has more than 0.5% of tuberculosis among its cattle. Much remains to be done to eliminate from our cattle the last remnants of the disease, but the loss, except for minor instances, is mainly over; the danger to the public health past. Nearly a quarter billion tuberculin tests have been made by veterinarians and almost four million reacting animals have been removed from the cattle herds of the United States. Every herd has been tested one or more times. Throughout the whole project, from the man in Washington who planned it to the man who tested the cattle in the dairy barn or on the range, the work has been in the hands of men who were educated to handle disease—men who knew the job they were expected to do. The condemned animals were appraised by veterinarians and paid for on their certification. The premises were disinfected under the supervision of veterinarians and the carcasses of the slaughtered animals were inspected by veterinarians. It was a grand job, and not a breath of scandal has attached to it from the beginning to the present moment. However, that record is not surprising; it is expected from any professional service.

★ ★ ★ BUY WAR SAVINGS STAMPS AND BONDS

Autobiography of a Veterinary Practitioner

By E. T. BAKER, Moscow, Idaho

X

BEGINNING PRACTICE

IN JULY, 1913, I began practice in earnest. I was just 30, with four years of diversified experience, and possessed good health. I do not think young men realize what a good field or a poor field means to them. A good location is where the people have enough livestock to support a veterinarian, and are accustomed to summoning expert help. Then, again, if the practitioners before him have been competent veterinarians and good citizens, the task is just that much easier. To locate in a community where the preceding practitioners have been unscrupulous drunkards or immoral scamps requires a good many years of "building up."

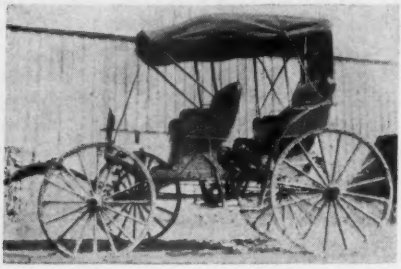
I had no car at that time, but hired one from the livery stable. They had an old auto—a Chalmers, I think, that had to be cranked—and it was an all day's job to change tires. It wheezed along the dirt roads at nearly 20 miles an hour and, considering the care it received, always performed nobly for me. In bad weather, or whenever it rained, the roads were impassable for the car, so a team and buggy was used. As I did all the veterinary work for the livery stable, our bills just about coincided, for I always presented my bill last.

The owners of this livery stable were two old brothers who had come west as boys in a covered wagon. They had homesteaded in the early days, and both were well off. They were lovers of good horses, and spent most of their spare time "cussing" the new fangled autos and trucks. They both passed on some 20 years ago, the last of a vanishing generation.

For $12 a month, we had rented a very comfortable house—one that, in this section, would bring around $35 today. Our first experience in renting was our last; as it turned out, we were fortunate in having a grasping landlord. When we moved into this place, it looked like a typical "renter house." The first thing we did was to purchase a lawn mower and some hose, and spend three or four dollars a month in watering the lawn and keeping it in good condition. For this expense and trouble, our landlord dropped over one beautiful afternoon and informed us the rent would be $18 a month from then on, as he could get that much for it now. We were so indignant, we started out to look for a house or vacant lot, and decided on our present location, where we built in 1916. However, we rented for several more years, and saved up enough to pay for our new home when we moved into it. The total cost, including pavement and various improvements, was around $6,500.

Perhaps this experience was the best thing that could have happened to us, for saving and sacrificing was a pleasure whenever we thought of how our landlord had charged us extra for improving his property. We have never been in debt since we were married, as we never purchase things until we can pay for them. By being patient, and buying a few essentials each year and paying for them, it is remarkable how soon one can have a comfortable home. Best of all, when one is out of debt, and hard

times come along, one does not have to worry about bills and wonder where the money is coming from. Being 75% Scotch has perhaps given me a thrifty streak, and due credit must also be given my wife, who always has been willing to sacrifice and



The type of vehicle used by the author in his early practice

to do without needful things until we could afford to pay for them.

My first year of practice, even with hiring "rigs," and being compelled to purchase some instruments and supplies, was very satisfactory, and much better than any previous salary. Paying cash for supplies gave me 10% off, so that in nearly 30 years I have had the equivalent of three years' supplies given me as a present. I made up my mind to devote all my energies and time to practice, and to avoid all get-rich-quick schemes or side lines. Adhering rigidly to this rule prevented me from taking advantage of three opportunities that came along soon after, which were as follows:

In the winter of 1913, I was called to investigate a disease among some horses employed at a zinc mine. My bill was around $150, and the superintendent of the mine, who was an old friend, urged me to take stock in lieu of money. Of course, no one knew that a world war would be on within eight months, and lead and zinc would zoom to the sky. Had I taken his advice, my $150 woud have amounted to over $3,500 in less than two years.

Opportunity No. 2 occurred in September, 1914, soon after the war had started. Two young Frenchmen, whose former homes had been in Alsace-Lorraine, offered me their entire outfit of 4000 sheep and all equipment for $10,000 cash, so they could return to France and fight. Not being in the sheep business, I turned the offer down, and another man purchased them, borrowing the money at the bank. By 1917, he had cleared over $60,000, besides having 6000 ewes worth $12 a head, and a year's crop of wool, which he was holding for 45 cents a pound. However, he overextended himself trying to get rich too fast, and, after the crash of 1919, when ewes dropped to $2 a head, and wool to half the cost of production, he lost everything he had, and is now back herding sheep at $40 a month.

My third financial lapse was made in 1915, when a good farm of 240 acres was up for sale to settle an estate. I could have purchased it for $60 an acre, borrowing all the money at the bank. In 1917 it turned for $200 an acre, and in 1918 for $225. In 1920 the last owner, who had paid $20,000 down on the place, gave it up as a bad bargain, and a few years ago it changed hands again for $75, its real value. However, I stuck strictly to practice, and figured it was never intended I be rich.

When our country entered the war in 1917, I immediately offered my services. However, due to a mixed up mess, which is so graphically described in Merillat & Campbell's "Military History," I never heard anything from the Surgeon General's office until the next June, when I was directed to proceed to Camp Lewis to take the examinations.

I was told by the examining officer, a regular army veterinarian, that I was the third general practitioner to appear before him out of a dozen who had been there before me. He said he hoped more general practitioners would show up, as they were urgently needed. One of the tragedies of that conflict was that meat inspectors and college professors were sent to doctor horses and mules while practitioners were detailed to inspect meat. Of course, during the later stages of the fracas this mix-up in personnel was somewhat rectified. Had the war continued after November 15th, I understand I would have been sent to Camp Greenleaf. A dentist who practices in my town assures me I didn't lose anything by not getting there. He wonders now if the

de luxe horse stable he occupied is still there.

Perhaps a war is a sort of revolution, and brings about changes that would not come otherwise. However, it takes a frightful toll of our best manhood and leaves the physically and mentally unfit to stay at home and procreate their kind.

We have always had wars and will probably have them until the end of time. As these words are written (1940) another and deadlier war is in progress. The outcome only the future can tell.

The average American did not prosper during the war. My practice was good, but the money rolled out just as fast as it came in. A few grew rich on the blood and tears of the victims, but it was a false prosperity, just as many people fight for false propaganda.

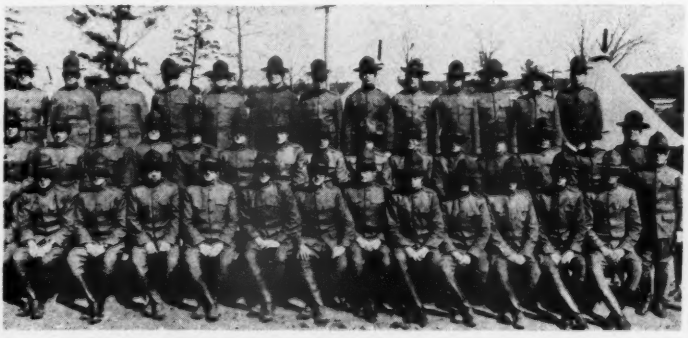
I had begun practice when two new eras were opening: First, the revolution in transportation and farm power; and second, the beginning of this great war, the effects of which are still being felt, even now that a second great conflict is raging. At that time I was young and did not take life very seriously, but older and wiser men shook their heads and said: "What goes up must come down." As an example of how hectic things were in those days, I remember being out on a call to a big wheat rancher's place. While I was still there, a grain buyer drove up and offered this farmer $2.80 for 20,000 bushels, or a check for $56,000. The owner held out for $3, but later sold the whole outfit for 90c a bushel; went broke and insane, leaving his family penniless.

To illustrate how good and bad fortune plays an important part in the destiny of man, I remember how two brothers purchased adjoining ranches in 1914, paying $9,600 for each quarter section. A few months later war broke out, and grain prices began to soar. One brother paid off his mortgage in three years and is now well fixed. The wife of the other brother contracted cancer, and he spent all his time and money trying to cure her. Several years later she passed away. This brother now works for the highway at day wages, and people new to the country wonder why he is not well off like his brother.

A country practitioner can explain many things like this. He sees the successes and failures, and knows of the joys and sorrows of his community as well as the country physician. Perhaps that is why practice makes one mellow and tolerant. He sees so much of human nature, and learns to take into consideration that uncertain quality known as "natural disposition."

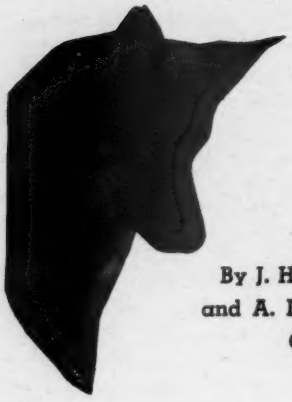
It may be compared to raising two heifers of almost identical color and breed. One turns out to be a good, docile milker; the other is a mean, vicious kicker, and does not pay for her keep. Perhaps you can explain it; I admit I cannot. In other words, why does a chicken cross the road?

(To be continued)



Veterinarians at Camp Greenleaf, Fort Oglethorpe, Ga., where the author was scheduled to be sent in 1918 had the war not ended suddenly. They are the veterinary officers of Company 27, 7th Battalion, Medical Officers' Training Camp

Clinical Coccidiosis in Puppies



By J. H. EMMERSON
and A. D. DASHOFF*
Quincy, Mass.

IN Greater Boston clinical coccidiosis is a common and highly fatal disease of dogs between the ages of six and twelve weeks. The majority die in convulsions. Symptoms are variable, determined probably by the species of secondary pathogenic invaders and the tissues involved. Only seldom is there fresh blood in the stool. This is due to the fact that the inflammation and ulceration is usually confined to the small intestine. On postmortem examination we seldom if ever find anything abnormal in the large intestine. Diagnosis is confirmed by microscopic examination, using the high power objective. Once the puppy shows symptoms of the disease (clinical coccidiosis) treatment is usually unavailing. We have tried about everything recommended in recent literature. It is best to advise euthanasia. Preventive measures are effective in controlling the disease; that is, routine fecal examinations of newly-purchased puppies, administration of bismuth preparations to positives, and the immediate disposal of fecal matter.

Symptoms.—There is always a diarrhea (usually no fresh blood) and often an elevated temperature. Champing of the teeth and frothing at the mouth are common. There may be a locomotor incoordination. Generalized convulsions indicate approaching death. A few develop chorea. Sometimes a puppy shows signs of colic—bloating, grunting pains on abdominal palpation, shrill howling, and biting hard objects. The animal may be anemic, emaciated, refuse food, or vomit. Dyspnea usually means that the lungs are involved. There may be pustular eruptions on the skin and a purulent or mucopurulent conjunctivitis at the same time.

On post mortem examination there is a definite ulceration and inflammation with scattered petechiae in the small intestine. The colon, cecum and rectum are usually normal. The ulcers permit intestinal bacteria to enter the blood stream and reach spinal fluid, giving rise to pneumonia, meningitis, and pustular dermatitis.

Diagnosis.—This is based on three factors: (1) One or more of the above symptoms; (2) Age: six to twelve weeks; (3) Positive laboratory findings.

It is essential to learn how to recognize coccidia. There are many veterinary textbooks describing the morphology of coccidia. The coccidia need not be confused with air bubbles, but there are a few important diagnostic facts not found in textbooks that we should like to mention. Usually the owner has administered oil, which may deceive the inexperienced. It is necessary to make examinations on three consecutive days before deciding on a negative diagnosis. One negative examination does not mean that the disease is not coccidiosis. The smear method is satisfactory, as are also the sugar and salt flotation methods. A slide may be loaded with coccidia and yet be overlooked with the microscope because of failure to employ the high power objective. The high magnification is necessary to make out the structure of the small spherical oocysts which are quite commonly found in dogs. The large egg-shaped oocysts are rarely found in this animal. Oocysts taken directly from rectal fecal matter seldom show division of the protoplasm into

*Since collaborating in this article, Doctor Dashoff has become affiliated with the Anaheim Dog & Cat Hospital at Long Beach, California. He reports that coccidiosis is a serious and often fatal disease in his state.

sporoblasts. It is always one central mass. The development of oocysts may be observed in feces liquefied with 2½% solution of potassium dichromate and incubated at 25°C. (room temperature). Sporocysts form after a few days and can readily be distinguished with a high power microscope.

Prognosis. — In clinical coccidiosis the prognosis is very unfavorable. It is best to advise euthanasia.

Top row, Spherical oocysts, non-infective stage (high power). 2nd row, Same oocysts (low power). 3rd row (left), Spherical oocysts (two sporocysts) infective stage (high power). 3rd row (right), egg-shape oocysts, non-infective stage (low and high power)

Treatment. — We have tried practically every remedy recommended, but with very little success.

Prevention of Clinical Coccidiosis.—Occasionally we are fortunate to have a puppy brought in for a physical examination or a worming and on fecal examination, find coccidia under the high power objective. These cases we do not consider clinical coccidiosis, but they are potentially clinical cases. They respond well to hygiene and bismuth preparations. We like the following preparation:

℞

Bismuth subnitrate .....ʒ j
Cerium oxalate .....ʒ ij
Phenol (liquefied) .....gtt x
Distilled water q.s.....ad ℥ ijss

M. et F. sol.

Sig. One teaspoonful T. I. D. for five to 15 pound dogs for 14 days.

It should be noted coccidiosis is rather common in kittens and can be mistaken easily for infectious enteritis. We have not attempted to treat such cases. They are destroyed.

Formula for Intravenous Use of Chloral Hydrate

The dose is 11 grams per hundred kilograms (5gm per 100 lbs.) of body weight of pure chloral hydrate. Designating by "P" the weight of the horse, the quantity of chloral to be used is deduced from the formula $C = \frac{P \times 11}{100}$; thus, for a horse weighing 500kg, $\frac{500 \times 11}{100} = 55$gm. Citrate of soda is then added at the rate of 50% of the weight of chloral, up to 30 grams. To make a 20% solution, the volume of water added would be C x 5; add to this one gram of sodium chloride per 125cc of water, to make an isotonic solution. The preparation indicated for an animal weighing 500kg (1100 lbs.) is therefore: 55gm of chloral; 22.5gm of citrate of soda; 2gm of sodium chloride, dissolved in 275cc of lukewarm distilled water. Dissolve the chloral hydrate in the physiological salt solution (not too hot, to avoid modification of the chloral); add the citrate of soda, finely powdered, and filter through cotton.

This solution should be made up fresh.

At this concentration the anesthetic solution is irritating for cellular tissue, and the injection must be strictly intravascular. It should be made against the flow in the jugular vein and should begin by holding the solution only 12 inches above the point of injection; allowing it to flow into the vein by gravity. When 250cc have been injected the bottle containing the solution may be elevated to 24 inches above the point of injection. A large needle should be used for the injection—not smaller than 12-gauge.

Many of our subscribers tell us that they "like to know what the other fellow is doing and how he is doing it"—you like to read of the other fellow's interesting cases; he also likes to hear of yours.

A Technic for Handling the Self-Sucking Cow

In the past, the self-sucking cow, like the windbroken horse, was something for the "other fellow" to own. Many devices have been tried from bull rings to all kinds of harness with little success. The operations performed by veterinarians were not always successful and sometimes were inhumane. The result was that many good cows with no other fault than that of self-sucking were sent to market or were sold at a price far below their value as milk producers. At present, and for the near future at least, our country needs all the milk it can produce and this waste should not be tolerated.

Any veterinarian can prevent a cow from sucking herself or other cows by a very simple operation. In fact, if the owner were not present when she was "cured" he would never suspect that anything had been done to the animal.

Credit for discovery must be given to Dr. Lee Marner, Wellman, Iowa. His technic is both simple and effective. Since not all veterinarians seem to make use of it, I will relate my experience.

The animal is placed in a stanchion or tied up short to a post. An assistant holds the cow's head up with a nose lead. The operator holds a hogringer in his right hand with a No. 2 (middle sized) hog ring in place. The cow's tongue is grasped and pulled out of the mouth with the left hand. One hog ring is placed about two and one-half inches back from the tip of the tongue on the ventral surface of the tongue exactly in the median line. This ring is placed across the tongue with the hog ring bisected by the median line. A second ring is placed in a similar manner about one and one-half inches behind the first. Tell the owner to turn the cow loose and forget about her. After a few minutes of useless trying to remove the ring the animal also will forget about it and will be reminded only at the particular time when she tries to indulge her vice.

The success of the operation lies in placing the rings so that cupping the tongue causes pain, as a cow must cup her tongue to suck a teat. As this is the only act of prehension in which she needs to cup her tongue, when the attached hog ring causes pain she soon decides that it is not worthwhile.

Doctor Marner advised two rings, however, I have found that one is sufficient and it should be left in permanently. I have never seen infection result from "ringing" the tongue; nor any difficulty in eating nor in drinking (after the first day or two) following this operation. The pain lasts but a second, and the use of local anesthesia is probably a greater annoyance to the animal than the operation. However, if local anesthesia is used it must be given sparingly, since any general anesthesia of the tongue is likely to result in its being injured by the teeth far more severely than the hog ring injures it.

I have used this technic on 30 animals successfully. Recently I put rings in eight yearling heifers all of which had developed the habit of sucking cows. They forgot both the rings and the habit almost immediately. In these 30 cases there was but one failure. The reason was because the owner became alarmed the next day when his cow did not eat and removed the rings.

Needless to say word will get around that Doctor Blank can break a cow of sucking herself by simply putting a hog ring in her tongue and owners attempt to copy the technic. Butchers tell me that they have found the ring in all sorts of locations in the tongue. Such ringing of course does no good as it does not cause pain when the tongue is cupped.

There are few owners who are not willing to pay the price of a call to make a producer out of an animal that is drinking up the profits. After one becomes adept at the technic no operation will prove more successful.

G. W. BAYLES

Marion, Illinois

★ ★ ★ BUY WAR SAVINGS STAMPS AND BONDS

Record Your Experiences

In speaking of the standing operation for castration, I was reminded of the article by Rembrandt Morgan of West Virginia, in which he said: "Tie 'em first."

Back about thirty years ago, when I was very young, athletic and active, I was called by an old-time vet to operate some cryptorchids. I was late in arriving at his place, and on our way to the first animal we were to operate, he stopped and said he had a straight colt to castrate at that point. It was in the early Spring and the tall, gaunt, half-starved two-year-old was standing in corn stalks up to his knees sunning himself. The owner saw us coming and turned to pump a bucket of water to bring to us. I jumped the fence with the emasculator under my arm, knife in hand. While Old Doc was pulling the throwing harness out of the back of the machine, I proceeded to castrate the colt. He swung his testicles well—I let them both out with a quick stroke. He humped his back and moved a step, and I crushed them both off. By the time Old Doc reached me, the animal was castrated. The owner came up with the water and asked where we wanted to throw the animal. The Doctor pointed to the testicles where they lay on the ground. The owner then asked me if I wanted to wash my hands, but I replied that I had no blood on them. It was a wonderful demonstration—had I gotten away with it but as I stooped to rinse the instrument in the bucket, I glanced up and there came the colt's bowels crawling down like an old snake. I was feeling pretty chesty until I saw that sight. Had I thrown the colt, I would have discovered that he had a hernia and thus avoided the embarrassing situation in which I found myself. Old Doc placed the hopples on the colt while I held the bowels, then we threw him —the bowels were returned and the colt made a recovery, which surprised us both.

With the use of proper restraint, I feel it is unnecessary to take chances of bumping into conditions such as Doctor Morgan and I have had. We get by wonderfully well for a long time, but sooner or later they catch up with us.

A few weeks ago, I was called in consultation with my good friend Doctor Archer of Terre Haute, on a fine saddle-bred mare that had been sent south and bred to a fine horse. The colt at her side was about six weeks old when she was sent south. After being bred the mare became very thin and dried up—the colt almost starved and had to be artificially fed. Later the mare began to strain. Upon examination the hand could hardly be introduced into the vagina, for there we found a recto-vaginal abscess, a tumifaction as large as a football, located between the rectal wall and the vagina. We operated this through the vagina and the mare is recovering.

I mention this because it is only the second case of the kind I have met with in almost forty-one years of practice. Both were operated alike and both made good recoveries. This condition was no doubt brought about at the time of copulation rather than during parturition.

At the AVMA section on surgery, quite a discussion took place on cancerous growths in the eyes of cattle. One question was asked: "In what breed is this seen most often?" In my practice I have found this condition oftenest in the Hereford. It seems to me that after many years in the field of practice, when we are consulted on a non-pathogenic, non-parasitic disease, we can right away name the breed. If we are called to a sore-eyed cow, we can generally ask if it is a white-face. I have had more trouble with injuries of the penis of Angus bulls than any other breed, but have met with more cases of growths on this organ in the Hereford bull. We do know that some animals have hereditary weaknesses.

I know that general practitioners are all busy, especially at this time, in preserving the health of our animal population, but I, like other practitioners, would like to read of the experiences of other men in the field of practice.

T. A. SIGLER

Greencastle, Indiana

Book Reviews

Ambassadors in White. A story of American Tropical Medicine. *By Charles Morrow Wilson.* Illustrated; 372 pages. Henry Holt and Company, New York, 1932. Price—$3.50.

This is an epic story of medicine in the American tropics. Of great interest at anytime, the subject derives a hundredfold increased interest at present when hundreds of thousands of America's armed forces are, or soon will be, stationed in these or other tropics.

A lead to the theme of the discussion is contained in a few typical statements from the texts: "Touring microbes like human tourists frequently misbehave upon arrival in the tropics. Latin American death rates from what we consider merely nuisance diseases—measles, chicken pox, etc., are several times greater than in the United States; pneumonia is more frequently fatal. Influenza kills its victims more than twice as often. Malaria is a positive plague, virulent, body blasting, so almost certainly fatal in wide districts, it is considered a waste of money to employ doctors or buy drugs." There are hundreds of examples of the ruthless intensity of Temperate Zone diseases transplanted to the tropics. Some of the increased virulence is explainable in terms of poverty, ignorance of hygiene and lack of medical and nursing care, but more of it is as yet unexplained. Microbes are still miles ahead of man in matters of adapting themselves to the tropics. The mysterious phenomena of climate which tends to make men listless and enervated in hot countries seem to give disease organisms added strength and inexplicable violence.

The population of Latin America is 120,000,000, of whom 50 million men, women, and children are constantly ill of serious diseases. It is a society of sick people. The meningitis rate in Chile is 20 times that of the United States, In New York City, the death rate from tuberculosis is 49 per hundred thousand; in Detroit, 48—but in Santiago, Chile, it is 430; in Lima, Peru, 435 and in Guayaquil, Ecuador, 693. Typhoid, dysentery and other water-borne diseases are actual menaces. Large areas in Brazil are reservoirs of yellow fever and the deaths from that disease have increased 1700% since 1908. Alarming outbreaks of typhus with a mortality of 33⅓% have been reported from half a dozen Latin-American countries within a year. European settlements were established in tropical America before they were in what is now the United States and with better prospects of succeeding. That they didn't keep pace with the colonization and development to the north of them is charged mainly to uncontrolled disease.

The author is optimistic as to the future of Latin America in matters of health. He cites examples to prove and quotes the opinions of qualified observers to the effect that an expenditure of only $3.00 per capita each year on public health would bring about health conditions in Mexico, Central and South America comparable to those in the United States.

Dr. Carlos Finlay, the Havana physician, Major Walter Reed and General William Crawford, medical officers in the United States Army, Dr. William Deeks, a Canadian physician and United Fruit Company doctor, all rate as top-flight heroes in the war on disease in the American tropics. A most delightful feature of the work is a biographical sketch of each of them. Their careers were more exciting than those attributed in fiction to filibusters and pirates.

Better disease control in Latin America would be of incalculable commercial advantage to the United States. It would, for example, provide us with all the rubber, quinine, palm oil, tung oil, tin and dozens of other products that we need. It would provide a reciprocal market for great quantities of American products. But most important to us right now is that we maintain garrisons and will perhaps even have field armies in contact with "the contagious, the polluted water supplies, the mosquito-ridden jungles, the fly-ridden borderlands and the contaminated milk and food" of the American tropics. The health problems of Latin America concern the United States most intimately. This work depicts them lucidly.

Breeding and Improvement of Farm Animals. *By Victor Arthur Rice,* Professor of Animal Husbandry, The Massachusetts State College, with chapters on the Endocrine Basis of Reproduction and Artificial Insemination, *by Dr. Frederick Newcomb Andrews,* Assistant Professor of Animal Husbandry, Purdue University. 750 pages, 272 illustrations, charts and graphs. McGraw-Hill Book Company, Inc., New York, 1942. Price—$5.00.

This is one of the most satisfactory texts in which a veterinarian can invest and surely the choice of the field on animal genetics. It is as far removed from texts of a generation ago on "breeds and breeding" as present-day works on nutrition are ahead of the books on "feeds and feeding" of the same period. The author states his aim to be to bring together in a single volume a complete course on reproductive physiology, genetics and the art of breeding, and to make it something more than a technical book on breeding by giving a hasty review of the background involved and indicating some of the broader implications of the place of man and his animals on this planet. In the opinion of the reviewer he has succeeded admirably.

The most unique section of the work, that dealing with the historical background of animal breeding, comprises 90 pages and in some way within that compass the author manages to deal with: animal breeding present and past, livestock trends in the United States, value of the average animal, value of purebred animals, foods of animal origin, breeding opportunities, the purebred era, rise of the breeds, medieval continental agriculture, Roman agriculture and animal husbandry, Grecian agriculture and animal husbandry, Oriental agriculture and animal husbandry, early man and animal domestication, early forerunners of man, early plant culture, the metal ages, domestication of animals, influence of domestication on civilization, origin and domestication of dogs, horses, cattle, sheep, goats and swine, and a chapter on animal origins and progressions —evolution.

Section II deals with the mechanisms of reproduction—the part of the male and of the female in reproduction, the endocrine basis of reproduction and lactation, fertility, reproductive efficiency, parturition, sterility and artificial insemination:

Section III in eight concise chapters discusses the mechanisms of heredity, including the history and problems of genetics, principles of heredity, principles of variation and sex determination.

And Section IV, the last, devotes 230 pages to the art of breeding—the application of the principles discussed in the preceding 500 pages of the text.

In the author's words: "the first section was designed to give the student an overall view of the history and status of breeding at the present time as well as an introduction to the origin and relations of all living forms in terms of the probable physical basis of life and inheritance—the genes and chromosomes.

"The second section deals with the scientific and practical phases of reproductive physiology in order to acquaint the student with the fundamental procedures in the production of a new generation and to indicate the practical procedures that will best ensure a high degree of reproductive efficiency.

"The third section deals with the workings of the genes and chromosomes in the perpetuation of similarities and the creation of new types among related forms.

"Such knowledge should enable the breeder to approach his practical problem of creating better animal types in a less cocksure manner and with a greater realization that he is dealing with a complicated mechanism which demands a knowledge of details as complete as it is possible to achieve. It may be argued that old-time breeders often achieved great success in total ignorance of the basic principles. This must be admitted, but we should be aware of the fact that less publicity has been given to those old breeders' more numerous failures. Their task was to create a few outstanding individuals, which they did; ours is to raise the average merit of all our animals, which can be accomplished only if we fully understand the natural principles underlying reproduction and the mechanisms involved in hereditary transmission.

"The fourth and concluding section, deals

with the practical applications of the general principles outlined in Sections II and III—the task of stimulating new and helpful variations and then holding the gains that have been made.

"Each breed of livestock, as now constituted, has as its genetic capital the complete reservoir of genes as they exist in the living members of the breed. Each breed has a certain number of good genes and a certain number of bad ones. If the number of good genes can be increased at the expense of the bad ones, the breed will progress. If the opposite result eventuates, the breed will retrogress."

Present-day students of animal husbandry and veterinary medicine have available this text. Students of former days have the choice of reading it or risk having their information on the subject surpassed by that of their younger confreres.

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The Springer Spaniel. *By Maxwell Riddle.* Second edition, 160 pages, 196 illustrations, 10 sire charts, Judy Publishing Co., Chicago, 1942. Price—$2.50.

The publisher tells us this is a complete presentation of the origin and development of the springer, an extended explanation of the standards by which the breed is judged and a full discussion of the springer in the show ring and as a field dog. Further that the 114 illustrations of leading spaniels and the 82 general illustrations of dog subjects are in themselves easily worth the price of the book.

The reviewer is inclined to agree as to the worth of the book to a springer spaniel fan. Unfortunately the discussion of the subjects mentioned makes up only some 75 pages and the text is cluttered up with an equal amount of discussion in which the author, a "breeder, judge, and journalist," is on less familiar ground. For example, a unique theory of heredity is propounded that will hardly lessen the reputation of Mendel. Hazards of in-breeding are attributed to the hypothesis that in such breeding desirable dominant characteristics continue at the source level but undesirable recessives pile up and eventually destroy all the good that has been accomplished by selection. On the subject of feeding this is contributed: "The heat of cooking kills any possible germ condition in the flesh"—and the author is a journalist. And again, "All the vitamins and mineral salts, which a vast quantity of vegetables can furnish, can be supplied in one or two feedings of condition powder with the dog's regular diet." Vitamin B_1 is credited with curing fright disease by supplying protein. To the general fund of information on canine distemper, this gem is added, "One attack brings immunity on the basis that the germ is in the blood and therefore a second acquirement of it does not disturb the system." Directions for photographing dogs are made equally meaningless by specifying the shutter speed without mentioning the aperture.

However meaningless the padding, when the author writes from experience he expresses himself understandingly, e.g., "The layman dog owner and the breeder who know most about the physical needs of the dog, are the first to patronize the veterinarian."

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Veterinary Surgery Notes. *By E. R. Frank,* B.S.A., D.V.M., M.S., Professor of Surgery, Kansas State College. Third Edition. Flexible leatherette spiral binding; planograph print, 255 pages, 106 illustrations. Burgess Publishing Co., Minneapolis, Minn., 1942. Price—$5.00.

When the second edition of this popular work appeared two years ago the hope was expressed that the next edition would appear in conventional book form, more lasting, more convenient and more fitting for sound text matter than the note form adopted. That hope is again ventured for the fourth edition.

The volume is primarily for veterinary students, to supplement or be supplemented by lectures, surgical exercises and clinical demonstrations. However, the practitioner will find it useful as a brief, concise exposition of all the ordinary and most other veterinary surgical operations. The illustrations are a particularly valuable feature. In many instances interest is added to the discussion by a brief report of the actual case illustrated.

The Officer's Guide. A ready reference on customs and correct procedures which pertain to Commissioned Officers of the Army of the United States. Ninth Edition. The Military Service Publishing Company, Harrisburg, Pa. Price—$2.50.

The automobile industry and countless smaller industries have received unstinted praise for the celerity with which they put their facilities into the mass production of war materials, after the attack on Pearl Harbor. But the book publishers really set the pace for conversion. The assembly line apparently started early December 8th and mass production of "War Books" has continued uninterruptedly. The Officers' Guide possesses an advantage among this avalanche in that the spade work for it was already done; eight editions having preceded this one. Probably for that reason, it is the most valuable and the least exasperating of the many guides for the neophyte in the army.

The revision includes all major changes to the end of June, 1942; in less important matters to June, 1941. Some parts have not been revised since the national emergency was declared—not even since World War I, e.g., this illuminating information occurs concerning "leaves": "A leave credit of one month accrues to each officer of the Regular Army on July 1st of each year." Be it remembered there are but 15,000 Regular Army officers in our army of 5,000,000. One may serve in most of our camps days and weeks without seeing one, and in most instances would not even know the officer belonged to the smallest contingent of the army, if he did meet one. Yet, except for sick leave and academic leave there is no other specification as to whom is entitled to leave in the whole volume. A weakness of the work is the many and extensive quotations from famous generals of the past and present. These are the sources from which to obtain information on tactics and strategy. The right officer to advise a newly-commissioned second lieutenant what to do when he reports at his first station is a first lieutenant, who has survived the same ordeal within a year, not a general who entered the army from West Point when the country was at peace, and conditions in no way resembled those of today.

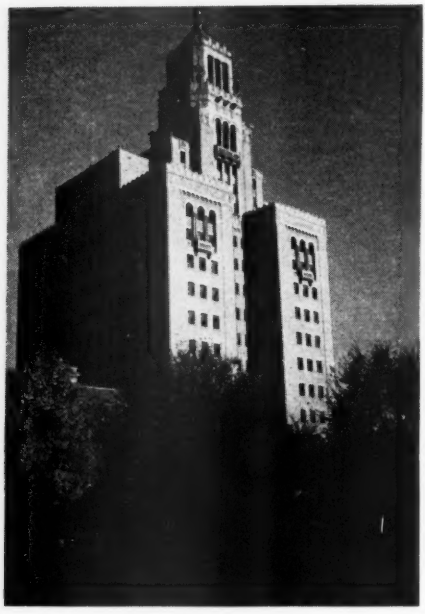
Emphasizing that the work is current, it begins with the President's War Message to the congress, December 8, 1941. The organization of the army—Ground Force, Air Force and Service of Supply, the Reserve Corps, the Officer Candidate Schools, the Women's Army Auxiliary Corps all are presented in their relations to one another. A good description is given of the Army's Educational System. The assignment of an officer to his first post, reporting, orientations, uniforms and needed equipment, command, administration, discipline, leadership, and management are discussed, as are also ceremonies, codes, customs, courtesies, criticism, cremation, privileges, pay, promotions, taboos, morale, leave, weddings, birth, retirement and the final will and testament. Public relations, even public speaking and writing home come in for attention. The aim obviously being to discuss every duty that may devolve upon the army officer. This object, however, is not quite attained, e.g., convoying. Many hundreds of officers are constantly engaged in convoying troops, yet the subject is not mentioned in this guide.

Notwithstanding its shortcomings, which in the main are those of including too much rather than too little, the "Officers' Guide" contains a great amount of information alike for the inexperienced and the experienced. It will constitute a useful guide and mentor to the officer from commissioning to retirement. It will aid in avoiding pitfalls and in military achievement. The purchaser will not regret his investment, as he may not find another single volume that covers the whole field of his duties so acceptably. It can and should, and doubtless will be improved in future editions, but there is not time to wait for them. This war, like all past wars, must be fought with what we have and this work will contribute its share.

You may obtain any book reviewed in this department by remitting the published price to the Book Department of VETERINARY MEDICINE, 7632 S. Crandon Ave., Chicago, Ill.

The Doctors Mayo. *By Helen Clapesattle.* 1942. 822 pages; illustrated. University of Minnesota Press, Minneapolis. Price—$3.75.

"An extraordinary book about three extra-ordinary men" will seem an extraordinary statement about the Doctors Mayo, for most readers, thinking of Doctor Will and Doctor Charlie, will ask "why three?" This work begins with the "Old Doctor," Père Mayo, truly a "horse and buggy doctor," when he was not afoot or ahorseback as he often was for lack of the buggy and



The Mayo Clinic

sometimes the horse. Dr. William Worrall Mayo was a pioneer in two states, and experienced all the hardships of a country practice on, and beyond, the frontiers of the 1850's. In every sense of the word the Old Doctor was a country doctor and he achieved not badly. His greatest achievement, of course, was preparing his illustrious sons to be country doctors for the nation.

Between them, father and sons, the Doctors Mayo lived a hundred years of American medicine and influenced profoundly its trend and progress during medicine's greatest century. At its beginning the stethoscope, the advertisement writer's symbol for "Dr.", was still novel, the clinical thermometer had not been invented, anesthesia was almost unknown, and blood counts were unknown. "Lung disease," "fever," "liver complaint," "inflammation of the bowels" and "kidney trouble" were erudite diagnoses. At the century's end there was—well, the Mayo Clinic.

The Doctors Mayo gives an intimate, fascinating picture, an authentic account of the lives of Doctor Will and Doctor Charlie. They were men who would have succeeded in any profession and in any location. That is made clear. But it does not explain in all its hundreds of pages their amazing achievement—why more than a million patients should have sought them from the ends of the earth, why "Mayos" should have become a household word all over America, how a village on the Minnesota prairie became a mecca for the medical profession to which the leaders from every nation on the globe journeyed for instructions, demonstration, inspiration. Perhaps the explanation cannot be given. Certainly the Mayos didn't know it and were as astonished as anyone at their fame.

Any comprehensive description of the Mayo Clinic, the crowning achievement of the Doctors Mayo would run into many pages. Perhaps the best description in a single paragraph is found in a letter written by former Secretary of the Interior Franklin K. Lane while a patient there. He wrote:

"Truly this is the most scientifically organized organization of scientists that ever was. Henry Ford could not improve upon it. Combine him with M. Pasteur, add a touch of Edison, and a dose of your friend Charlie Schwab, and you have the Mayo Clinic, a big, systematized, modernized, machinized doctorial plant, run by a couple of master workmen. . . . Tomorrow I am to be photographed and fluoroscoped—and then will come the verdict. . . . I guess whatever is said will be the last word—the Supreme Court decision. Fine reputation, that for two young chaps who never went to Harvard, eh what?"

Of the many biographies of medical men that have issued from the presses recently this book, "The Doctors Mayo," deserves first rank.